THE Review of Gastroenterology

VOL. 20, NO. 11 NOVEMBER, 1953

Internal Hernia

Diseases of the Small Intestine

Physiopathological Changes in Upper Gastrointestinal Bleeding

The Effects of Pneumoperitoneum Upon the Digestive Tract

An Evaluation of Prantal in Ulcer Management

Case Report—Multiple Duodenal Ulcers in a Nine Year Old Boy

Nineteenth Annual Convention
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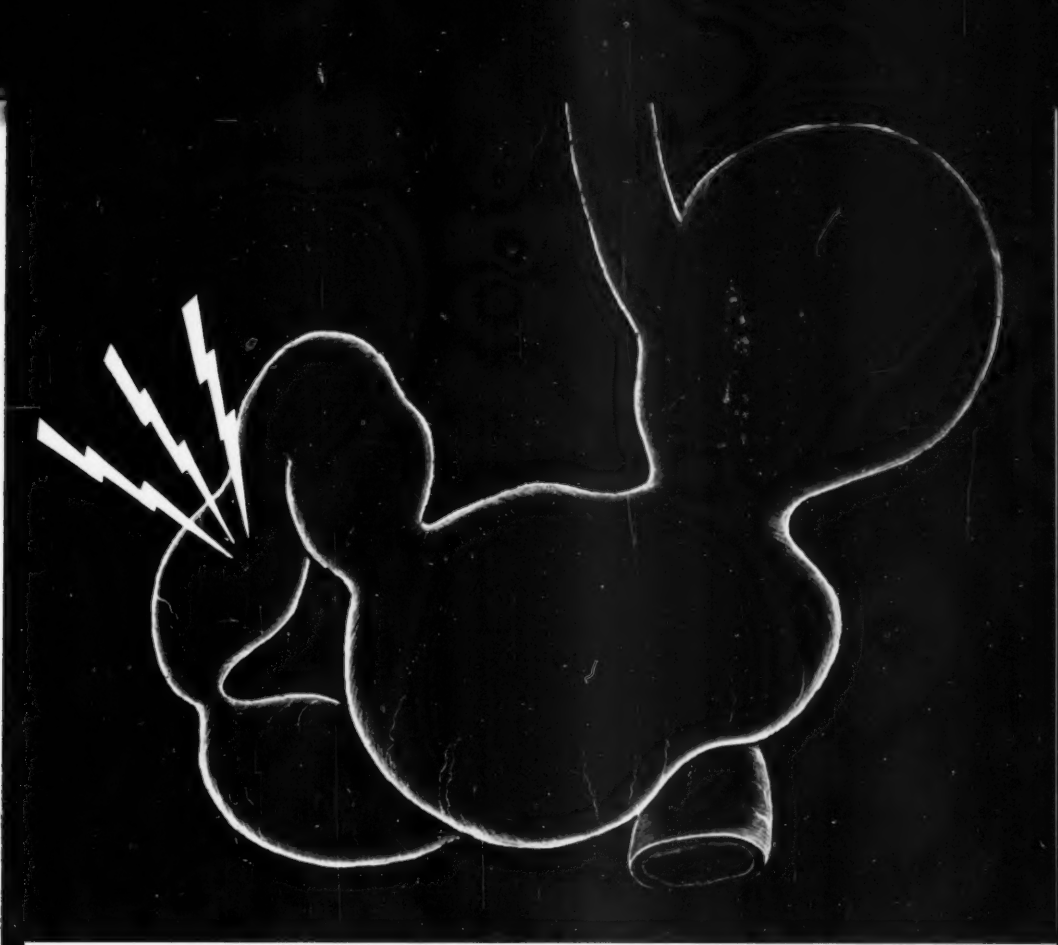

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1. Schwartz, I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: A Clinical Evaluation of a New Anticholinergic Drug, Pro-Banthine, to be published.

2. Ruffin, J. M.; Baylin, G. J.; Legerton, C. W., Jr., and Texter, E. C., Jr.: Mechanism of Pain in Peptic Ulcer, Gastroenterology *23*:252 (Feb.) 1953.

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THE Review of Gastroenterology

(INCORPORATING THE AMERICAN JOURNAL OF GASTROENTEROLOGY)

The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

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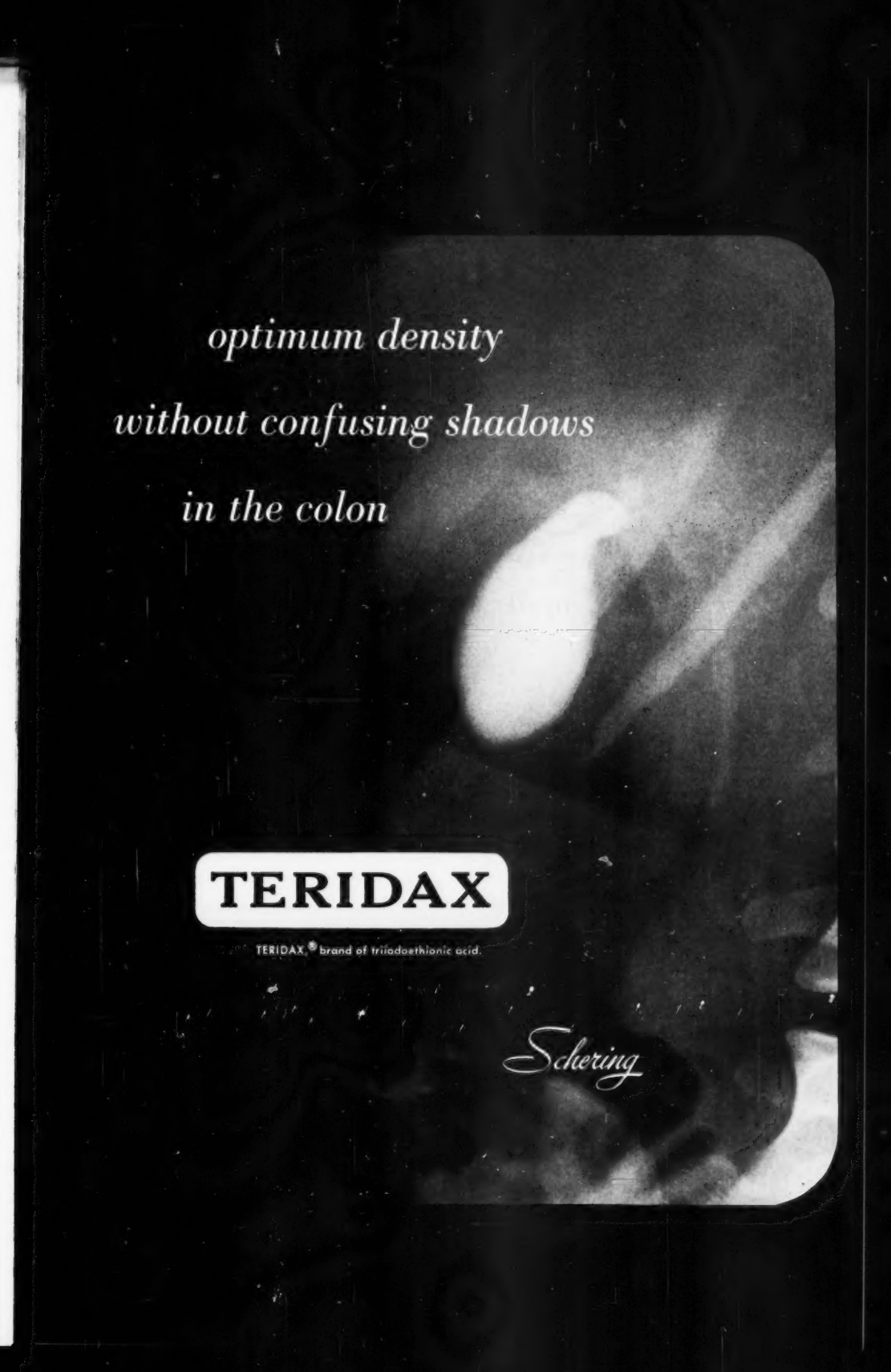
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1. Rev. Gastroenterol. 19:660, 1952
2. Exper. Med. & Surg. 9:90, 1951
3. J. Philippine M. A. 26:155, 1950

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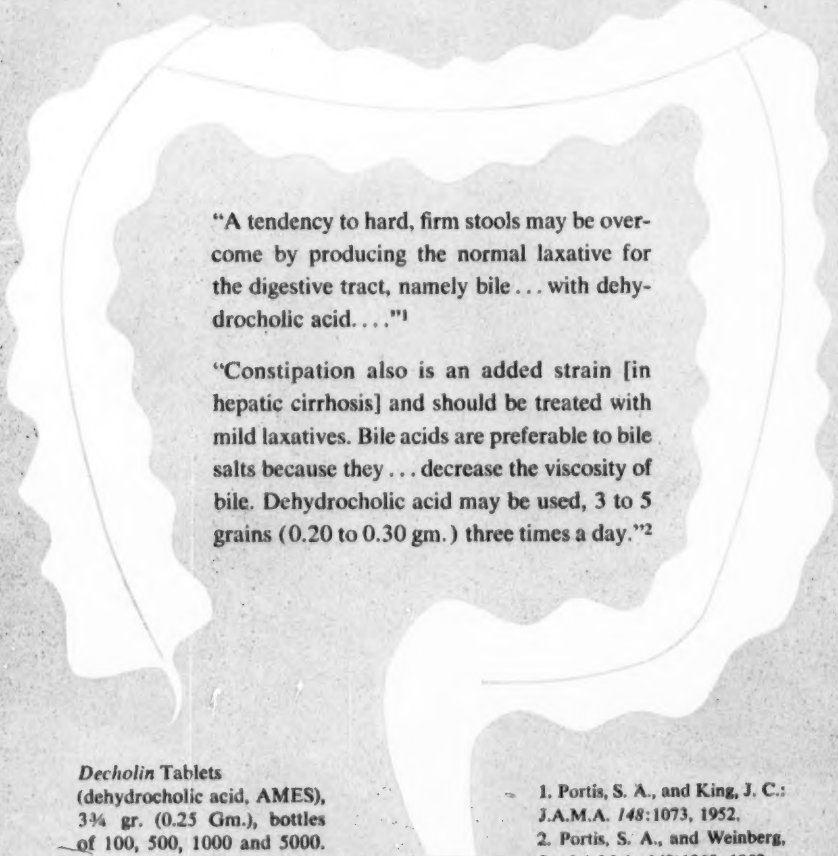

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1. Portis, S. A., and King, J. C.: J.A.M.A. *148*:1073, 1952.
2. Portis, S. A., and Weinberg, S.: J.A.M.A. *149*:1265, 1952.

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THE Review of Gastroenterology

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(INCORPORATING THE AMERICAN JOURNAL OF GASTROENTEROLOGY)

VOLUME 20 NOVEMBER, 1953 NUMBER 11

INTERNAL HERNIA*

HELMUTH NATHAN, M.D.
and
JOHN E. MOSELEY, M.D.
New York, N. Y.

A hernia is a protrusion of any viscus through an opening in the cavity into which it belongs. An internal hernia is a protrusion within the abdominal cavity. The herniation may occupy a cavity or fossa normally existing and pathologically enlarged, or it may take place in abnormal defects or sac formations.

The internal hernia is a rare condition. The famous English surgeon Moynahan has stated more than 50 years ago that "it does not find the attention in the medical literature which it deserves". Fifty years have passed since Moynahan's statement. Still, looking through the great textbooks of Surgery and Gastroenterology, we find this chapter not treated at all or often negligently surveyed. It is a subject of more than pure academic interest. The internal hernia may cause intestinal obstruction or strangulation. The diagnosis is difficult; the mortality rate because of frequent delay of operation very high. Surgery necessitates exact knowledge of the normal and pathological anatomy, especially of the difficult vascular structure to avoid irreparable damage.

Our chairman, Dr. Wangensteen, in his excellent textbook on Intestinal Obstruction, again stresses the point that the internal hernia is found infrequently. In a thorough review Charles C. Mayo reports his few experiences and notes particularly the rarity of the internal hernia.

In contrast with these statements are a few reports of pathologists and anatomists who found enlarged folds as potential hernial sacs in a high percentage of cases examined.

*Presented before the Course in Postgraduate Gastroenterology of the National Gastroenterological Association, New York, N. Y., 23, 24, 25 October 1952.

It is an amazing experience, looking through the casuistic reports here and abroad, to see the names of an unusual number of well-known men connected with this interesting condition. Grawitz and Haller, Moynahan and Mikulicz, Aschoff, Sonnenberg, Esmarch, Lahey, Coley, Wangensteen, Mayo and others have reported their own experiences on this condition. Once their attention has been focused on this subject, most of them have seen another or even more cases. Our own experience moved in the same direction: one case made us interested—subsequently we found a number of patients affected with this pathology. Altogether we have seen 7 cases of paraduodenal hernia alone and reported 5 of them previously.

Fig. 1—Subtotal gastrectomy. Inadequate closure of the gastrohepatic space of the mesocolon with the gastroanastomosis.

I hope that we inspire you and that next year we will hear several casuistic reports on this subject from this platform. A short historical review is justified: Hensing was the first to describe the fossa paraduodenalis (1742). Monro Haller (1803), Sandfoot (1786), Huschke (1844) followed with anatomical studies. In 1857 Treitz wrote his excellent thesis not only on the anatomy with the description of the suspending ligament, but he also mentioned the fold at the duodenal junction which carries his name, and describes the vascular arch. Further studies were made by Gruber (1859); our knowledge was increased with the description of another fold. Waldeyer and Toldt followed. Broesicke, in an excellent but pedantic study, discussed the anatomical condition and embryology (1891). In 1897 Moynahan's famous "Arris and Gale" Lecture appeared, with the perfect clinical study of the anatomical embryological and pathological

findings. In 1921 the first x-ray visualization of an internal hernia was made by Kummer. Andrews in 1923 gave new ideas to the development of the hernia and found an embryologic explanation of its cause. In 1933 Exner introduced the x-ray examination of the internal hernia and gave a review of the examination of 25,000 autopsies with 5 paraduodenal herniae. Alexander in 1937 improved the x-ray studies presenting a number of new cases. In 1941 Mayo gave his comprehensive review of this type of hernia including not only the paraduodenal but also the other types of internal hernia. There are various types of internal herniae.

Mayo speaks of those in normal pockets:

1) Foramen of Winslow
   Paraduodenal
   Paracecal
   Intersigmoidal

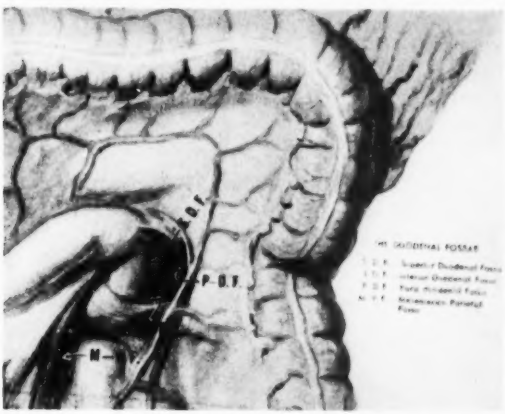

Fig. 2—Anatomy of duodenal fossa. S.D.F.—Superior duodenal fossa, Broesicke. I.D.F.—Inferior duodenal fossa, Treitz. P.D.F.—Paraduodenal fossa, Landzert. M.P.F.—Mesenteric parietal fossa, Waldeyer. (*Courtesy Ciba Pharmaceutical Products, Inc.*)

2) Those in exaggerated normal folds, broad ligament, etc.
3) From traumatic or operative bands
4) Chronic inflammation
5) In anatomic defects

A simplified classification may be:

a) Postoperative
b) Pathologically enlarged folds and fossae
c) Malformation
d) Miscellaneous

Wangensteen classifies the internal hernia as:

1) Diaphragmatic hernia
2) Hernia of the foramen of Winslow
3) Hernia of the paraduodenal fossa
4) Persistent hole in the mesentery
5) Hernia into the intersigmoidal fossa
6) Pericecal hernia

I feel that we may exempt the diaphragmatic hernia from our group: (a) For technical reasons, (b) the diaphragmatic hernia protrudes outside of the abdominal cavity and is not an abdominal hernia in the proper sense. Although the

Fig. 3—Left paraduodenal hernia. (Author's case.)

postoperative hernia is only accepted by few authors, we feel justified to include a certain group of them in our lecture.

The postoperative hernia can easily be understood. It is most frequently found following gastric surgery. An insufficiently closed mesocolon around a gastroenterostomy or gastric resection may allow the small intestinal loops to enter the lesser sac and to become obstructed. It occurs rather frequently when a posterior retrocolic gastroenterostomy is made and the loops were taken too long. Even with the closure of the mesocolon new deep fossae may be formed next to the jejunal anastomosis. The sutures may re-open and the same condition may exist as described above. The symptoms may develop immediately after the operation, but sometimes they are manifested later. They are not necessarily characteristic of a strangulation, but they may be like an intermittent incarcera-

tion, or of vague chronic nature. X-ray films may be very difficult to evaluate; they will often show signs of a high intestinal obstruction with dilatation of the stomach stump. Fluid levels are seldom seen because of frequent vomitus in these thirty cases removing the fluid in the upper tract. Internal herniae may also develop when a double loop colostomy was made with long insufficiently adapted loops or, if any form of intestinal resection with side-to-side, or side-to-end anastomosis, where pockets at the anastomosing stumps may easily remain. Good peritonealization is mandatory. Gynecological operation with incompetent peritonealization, anteflexion operation with careless closure of the defect in the ligamentary attachments can sometimes be blamed for the hernia formation. The Baldy Webster operation is especially known for this complication. The

Fig. 4—Right paraduodenal hernia, strangulated loop of ileum at distal opening of hernial sac. (Author's case.)

Nobel operation is advised for the prevention of adhesions and recurrent obstruction. We have seen one case where the segmental Nobel operation was performed and a fossa was formed at the point of junction allowing the intestinal loop to squeeze itself in. Recently we saw a patient in whom a previously mobile cecum had become attached to an abdominal scar and formed a hole through which intestine had entered.

The second group of internal herniae has already been described by Moynahan.

Nine fossae can be found around the duodenojejunal junction:

1) Fossa superior duodenal

2) Inferior duodenal (Treitz)
3) Paraduodenal (Landzert)
4) Mesenterico-parietal (Waldeyer)
5) Mesocolic
6) Posterior duodenal (Gruber)
7) Duodenojejunal
8) Recessus intermesocolicus transversus
9) Infraduodenal

While fossae herniae were known to occur in the first four, no hernia in the last five fossae has been described yet.

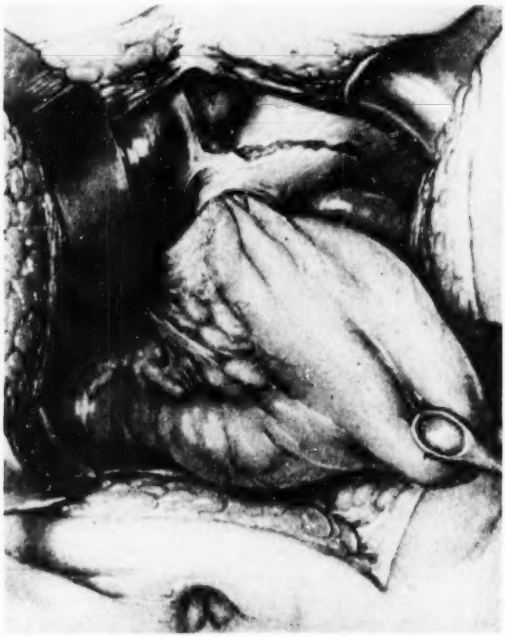

Fig. 5—Paraduodenal hernia from: *Watson, L. F.: "Hernia", C. V. Mosby, Co.*

Around the cecum we have the following fossae:

1) Ileocolic
2) Anterior vascular (Luschka)
3) Accessory anterior vascular
4) Ileo-appendicular
5) Ileocecal infi
6) Retrocolic-retrocecal
7) Iliac-subfascial
8) Retrovermiform

In the sigmoidal area:
Fossa retrosigmoidalis

In the vesical area:
Retrovesical fossa
Prevesical fossa

As malformations there are holes described in the different attachments of parts of the intestines.

Mesocolic
Mesenteric } holes
Mesoappendix

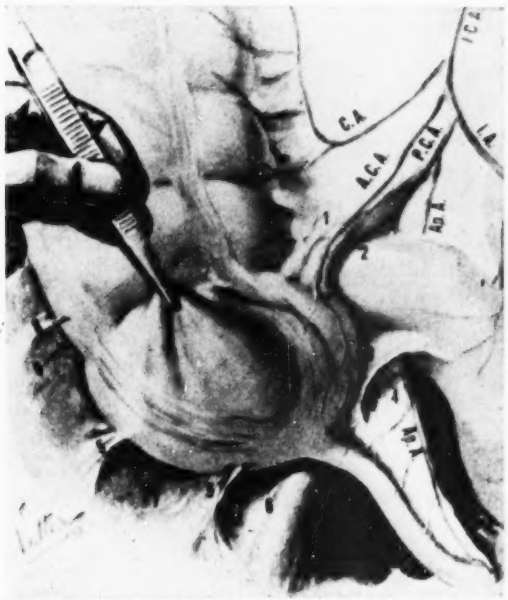

Fig. 6—1, 2, 3, 4, Ileocecal fossa; 5, Retrocecal folds; 6, Medial retrocolic fossa; 7, Retrocecal fossa; 8, Retrocolic fold; 9, Lateral retrocolic fossa. (*Courtesy Ciba Pharmaceutical Products, Inc.*)

Fossae around ligaments:
Falciforma hepatis,
broad ligament

A number of theories have been advanced to explain the herniation of fossae and folds near the doudenum. Treitz attributed the herniation into these fossae to a combination of tense margins of the fossae, freedom of movement of the intestines and peristalsis. Waldeyer considered the elevation of the peritoneum by blood vessels an important factor. Toldt explained the fossae as physio-

logic adhesions. Other theories gave the following causes: embryonic origin, where the anterior fold with its attachment to the anterior wall, and the posterior fold attached to the posterior wall concur with the development; late descent of the cecum; formation of pockets during intestinal rotation; formation of fusion folds in fetal life and failure of the root of the mesentery to unite with the posterior abdominal wall. Moynahan, Langer, and Toldt, Lower and Higgins and others expressed agreement with the view that the fossae into which herniation takes place are formed by fusion folds. This theory, which was accepted for a long time, was rejected by Edmund Andrews, who considered the term "duodenal hernia" a misnomer and gave an embryologic explanation of its causation.

Fig. 7—Sigmoidal hernia. The ileum is seen incarcerated in the hernia. Direction, medial-lateral.

He called the condition "a congenital anomaly due to imprisonment of the small intestine beneath the mesentery and the developing colon." He expressed the belief that the prevalent conception, that these herniae originate in small peritoneal pouches and grow in size by gradual expansion of the sac, and that the hernia is a pouch of abnormal origin, is absurd and grotesque. This he supported with the following statements:

1) *Differential pressure is utterly lacking* within the abdomen. Any vis a tergo to account for the growth of such herniae is entirely absent.

2) There are *hundreds of similar folds and fossae in the peritoneum,* many of much larger size, which are practically never the sites of such herniae.

Among the internal herniae the entire group collected under the name of hernia of Treitz is the most frequent one. It appears in about 75 per cent of all

cases. A recapitulation of the anatomy may be justified: The ascending part of the duodenum enters the peritoneal cavity and from the point of entrance we have the jejunum which is suspended with a ligament underneath the mesocolon transversum. This ligament is called the ligament of Treitz. On the left side we find a long semicircular fossa formed by a peritoneal plica coming from the descending colon and running in an arch to the transverse colon to the height of the pancreas. On the medial side of this plica either directly at the edge or—and this is important for surgery—up to 1.4 cm. inside of the edge, runs a large inferior mesenteric vein which is frequently accompanied by the ascending branch of the left colic artery. This plica covers the Landzert fossa, while the superior fossa carries Brosicke's name, and the lower one that of Treitz. Medial to

Fig. 8—Prevesical hernia.

this fossa we have the descending part of the jejunum, and in back the area of the second lumbar vertebra. A hernia which would develop here would show the opening of the sac toward the right side, and the enlargement of the hernia would protrude towards the medial and lower part of the peritoneal cavity. On the right side of the jejunum we have the other important fossa carrying Waldeyer's name, which in its plica has the superior mesenteric artery. This vessel is as close to the edge as said above of the inferior mesenteric vein. A hernia from this side could develop in two different directions: either retromesocolic pushing the right colon far laterally or far medially, or it could protrude anteriorly with an opening to the medial side. The larger the hernia becomes, the more will the opening project downward and posteriorly.

Summarizing: the genesis of the hernia was differently explained by Broesicke, Jonnescu and Moynahan. They thought that the incongruence of inside pressure of the jejunum and possible peristalsis of its wall caused a protrusion of the jejunum underneath the plica and a formation of a hernial sac. Andrews considered the adhesions to be derived from retroperitoneal tissue rather than due to inflammation, and blamed malrotation and malfusion in the embryonic period of life for this pathologic condition. The fact that a hernia was found in a new-born seems to support this theory which was accepted by Dragstedt, Wangensteen, Coley and others.

In the group of paraduodenal herniae the left duodenal hernia is the most frequent one. It appears in about 53 per cent of all internal herniae. The next

Fig. 9

Fig. 10

frequent one is the right duodenal hernia, of which there have been only about 50 cases reported. While the opening of the left hernia faces the right side and usually contains the entire small intestine, the right hernia opens to the left side and may contain only a part of the small intestine as seen on these two pictures: one taken from Wangensteen's book, the other from one of our own papers. You see the difficulty you may encounter when you have to perform a resection. The gangrene of the jejunum will descend further than the incarceration and strangulation occur because the vascular damage will be within the sac. The condition for an anastomosis just below the ligament of Treitz will not be favorable because of the difficulties of proper blood supply. This leads us to the clinical symptomatology. I am presenting a patient today who has the most beautiful x-ray findings you can wish for. He is a photographer who for many years has been treated for

an ulcer duodeni. After several years of living and fighting in Israel he came to this country. His complaint was pain which rarely appeared after eating, more frequently after drinking beer or any large amount of fluid, and especially when he is in a run-down condition. His gastric chemistry values are normal; he has no occult blood and no signs of intestinal obstruction. There is no indication for surgery unless he has real symptoms of obstruction when surgery should be performed immediately.

In most cases the degree of herniation has been a total one. Other patients may have symptoms of high intestinal obstruction—sometimes mild in form—sometimes leading quickly to an acute strangulation which makes immediate surgery necessary. After intake of small amounts of fluid the patients start to vomit at once, and obtain quick relief after vomiting. It is amazing that these patients may previously have been operated on, and that the surgeon does not notice any pathological condition.

Occasionally the first symptoms appear following an operation in the lower abdomen. The intestines, as usual, become paretic during the first postoperative days. As soon as peristalsis starts, the patient develops the clinical symptomatology of strangulation ileus. X-ray studies are the most important help in the diagnosis. High intestinal obstruction postoperatively should make us think of the possibility of an internal hernia.

Another clinical symptom is the pain coming and going with the intake of food. The lower abdomen may be soft, while the epigastrium is hard and tender. Vomiting again gives immediate relief from the acute pain. Often a tumor can be palpated. X-rays will be helpful in the examination. Those findings will be discussed by Dr. Moseley.

In doing surgery we must always think of the following two facts: in the right paraduodenal hernia the superior mesenteric artery runs along the anterior free margin of the sac. In the left paraduodenal hernia the inferior mesenteric vein can be found in the free margin of the anterior wall.

Surgery should be done under consideration of the standard rules of operation on obstruction. The sac should be exposed, if possible laid open under careful preservation of the vessels. Injury to the vessels may mean death! The sac may be inverted with interrupted sutures of the Stewart type, and the mouth of the sac fixed to the posterior wall, carefully preserving the vessels. In 1923, the mortality rate was still 90 per cent, in 1932, 20 per cent. We have performed surgery in 6 cases, with no mortality.

The next important group are the herniae in the ileocecal area. Here we have, as in the retrosigmoidal area, deep fossae which can easily become hernial sacs.

The mesentery of any part of the intestinal tract may have a hole in which intestinal loops may become incarcerated. Fifty cases of this type of hernia can

be found in the world literature. In autopsies 3/1000 are found. At times they are small enough not to cause any symptoms.

A very interesting type of internal hernia is that of the Foramen of Winslow. A common mesentery of the intestinal tract is necessary to make the loops mobile to enter through the foramen. In one case the incarcerated colon found its way out of the lesser sac into the free cavity again. The symptoms are those of an intestinal obstruction. The stomach is usually pushed anteriorly and laterally to the left side. A rare combination of a very high and low obstruction may be apparent. At operation it may be difficult to pull the loop out of the cavity, and it is advisable to open the lesser sac. If after emptying and decompression of the loops reduction of the hernia is not possible, resection is advisable. The mortality of this type of hernia is still 80 per cent in the strangulated cases.

Intersigmoidal hernia because of its rarity is difficult to diagnose. Its appearance can be seen in Figure 7.

Supravesical internal herniae, true intraligamentary and false median herniae are very rare, causing symptoms only in case of incarceration and strangulation.

Summary

Internal herniae are rare but important clinical conditions. At times they cause vague clinical symptoms and are recognized incidentally. Sometimes they cause intestinal obstruction. Whenever a patient has signs of intestinal obstruction without previous surgery, we must consider the possibility of an intestinal hernia, although this *may* occur after previous surgery.

All gastroenterologists and surgeons should be familiar with the anatomy, clinic and roentgenology of the internal hernia.

When no symptoms of obstruction are present, the patient may be treated conservatively but should be informed of the potential danger and the necessity of surgery if symptoms appear. The high mortality can be reduced by early treatment. To think about it is the most important duty.

The basic facts of the anatomy, clinical symptomatology of the various types of internal hernia, are described. The importance of early recognition is outlined.

Discussion

Dr. John E. Moseley:—The part played by the radiologist in the preoperative diagnosis of internal herniation is more or less confined to those cases in which the patient presents himself with rather vague symptoms of abdominal distress, or symptoms suggesting the possibility of a peptic ulcer. In these cases the patient is referred to the radiologist for a routine gastrointestinal series, and the filling of the gastrointestinal tract with barium mixture permits the visualization of the herniation. In those cases, however, in which the herniation presents as a com-

plete or incomplete obstruction, the radiological examination is usually confined to flat films of the abdomen, and the radiologist usually offers only a diagnosis of mechanical obstruction without attempting to define the nature of the obstructing lesion.

As Dr. Nathan has pointed out, this is a rather rare condition. I have reproductions from three cases.

(Film) Routine gastrointestinal examinations as usually done consist of a fluoroscopy of the esophagus, stomach, duodenal bulb, and first and second portions of the duodenum. After studying the esophagus, stomach and duodenal bulb, the examiner then looks for disturbances of the mucosal pattern in the descending portion of the duodenum, and is concerned with the form of the duodenal curve. After that the examination is stopped and films are taken, and the patient is not reexamined until after six hours, at which time the barium is in the terminal ileum and possibly down to the descending colon.

In order to make a diagnosis of herniation of the small bowel, it is almost mandatory that the patient be examined between the first and sixth hour, periodic examinations of the small bowel being made hourly. It is, therefore, important to consider the possibility of an internal herniation before these examinations are begun.

This is the patient Dr. Nathan mentioned to you who was treated for several years for peptic ulcer of the duodenum both here and in Germany. At no time was the possibility of internal herniation mentioned. On this examination, however, at the first observation the stomach was outlined and the duodenal bulb was seen to fill normally.

(Slide) This is about what the fluoroscopist would see, the duodenal bulb and a portion of the proximal duodenum. That is all we can see at this point on the first film (Fig. 9).

However, one hour after the ingestion of the barium, numerous loops of small bowel are seen to be confined somewhat to the left of the midline, and very sharply circumscribed in the area as if confined within a sac. The loops are in close proximity to each other and pressure by palpation on this collection of small intestinal loops would not disperse them as would ordinarily occur in the normal small bowel.

(Slide) At three hours this conglomeration of small bowel loops, again very sharply outlined and circumscribed, is seen displacing the stomach cephalad. This is the typical picture of a left paraduodenal hernia. It can be differentiated from normal small bowel by the fact that the outline of the herniated loops is so sharply delineated, and the fact that pressure does not separate these loops or change the rounded configuration they assume within the sac (Fig. 10).

The next 3 slides are those of a case reported by Reeves, Moran and Jones of the Department of Radiology of Duke University.

(Slide) This is of a 28-year old man who had five different attacks of acute abdominal pain in the course of two months. These were marked by sharp crampy pains, periumbilically, associated with vomiting of food ingested during that day. He was treated each time by hypodermic injections and enemas, which seemed to relieve him. After the fifth attack, the patient was given a gastrointestinal series, and the barium followed down through the esophagus and through the stomach, and the first portion of the duodenum. It was then seen to traverse the descending limb of the duodenum, but was not seen to continue over in its normal anatomical course across the abdomen to the left side, but rather was seen to dip down on the right side where most of the small bowel was then found.

Here again you will note a very sharp delimitation of these loops of small bowel which are very close in proximity and which cannot be dispersed by pressure. In a routine fluoroscopy, should the barium be seen coming down on the right side, one should immediately consider the possibility of either a malrotation of the small bowel or internal herniation. That should be enough to suggest following the barium through the small bowel by periodic examinations between the first and sixth hours, and during that time, when the patient is fluoroscoped, pressure can be exerted on these loops to determine whether they can be dispersed on not.

(Slide) This is the same patient six hours after the ingestion of barium. The important thing to see here is that the terminal ileum has been depressed, displaced downward, and the ascending colon has been displaced laterally. The transverse colon has been displaced upward, and the colon and terminal ileum tend to conform to the presence of a large mass in the abdomen a little to the right side.

This is a right paraduodenal hernia, as opposed to the first case which was a left paraduodenal hernia. These are the two most common types of internal herniation diagnosed radiographically.

(Slide) This patient was operated upon after that finding, and a right paraduodenal herniation was found. This film was taken postoperatively to show the return of the small bowel to its normal position in the abdominal cavity.

The next 3 slides are from a case reported by G. H. Alexander and H. H. Richardson from the U. S. Veteran's Hospital in Aspinwall, Pa.

(Slide) This case is that of a 52-year old man who came to the hospital in a condition of acute intestinal obstruction. He had had no previous gastrointestinal complaints, and he was treated conservatively by a small bowel decompression, and recovered on that conservative therapy. He was returned to the outpatient department for a barium meal examination, and at that time, the first film showed barium coursing normally through the stomach and duodenum, but superior and posterior to the stomach there was a collection of barium in a loop

of small bowel. Associated with that was a small, semi-circular collection of gas. I don't know whether you can see that rarified shadow just above the barium; however, most of this small bowel was hidden by the filled stomach.

(Slide) On a film taken two hours later, however, the barium was still seen to be present in the knuckling loop of small bowel, in the region of the ligament of Treitz. Again some gas was seen in the uppermost portion, and this peculiar loop was fixed and stayed in that position on palpation. One might suspect the probability of some type of herniation.

(Slide) At the three hours' observation, after most of the barium had continued distally in the small bowel, a small collection of barium was still present in that region and now a larger collection of gas, which apparently was being trapped in the herniated loop of small bowel is seen.

(Slide) At the six-hour observation, when the intestine had moved in its position, this loop of bowel persisted in the area exactly where it was seen on the first film. The barium had been emptied, but there was an increasing amount of gas in that loop, and the diagnosis of an internal herniation was made preoperatively. The patient was operated upon, and a herniation through the transverse mesocolon, in the region of the ligament of Treitz, was found.

The preoperative diagnosis, radiographically or otherwise, of internal herniations, is not commonly made, but it is terribly important to keep in mind the possibility of this type of lesion with just vague abdominal complaints, because if that is not done, the usual type of routine barium meal examination will skip most of the small bowel. It will show the stomach and first and second portions of the duodenum, and take up again, at the six-hour observation, and an internal herniation of the small bowel can be completely missed, as it was for so many years in the first case.

Dr. I. Snapper:—It is fortunate that these congenital internal herniae with incarceration are rare occurrences. As Dr. Nathan has underlined, the most important point in the recognition of the internal hernia is the realization that the neck of these paraduodenal herniae does not only consist of membranes or adhesions, but also of an artery or vein, which under no condition should be ligated.

Dr. O. H. Wangensteen:—No one really gets to be an authority on some of these rare internal herniae that were mentioned in this discussion. Schuh, a colleague of Billroth, said many years ago that a man could well spend his life studying the matter of hernia. That was before the modern operations of Bassini and Halsted. Yet the mortality of strangulated external hernia is still very real.

One can see at once the enormous disadvantage that a patient is subjected to in the recognition of the presence of an internal hernia. When a man has a pain and swelling in the groin, and tenderness over it, it is axiomatic that there is something there that should have surgical attention. In the abdomen, the problem is not so simple. In the first place, it is a more insidious sort of affair because

of the circumstance that the strangulating mechanism is rarely as rigid. Think of how rigid the structures are in a strangulated femoral hernia . . . Gimbernat's ligament medially and Cooper's ligament below and Poupart's ligament above. All these are unyielding structures.

Well, these circumstances rarely obtain in the peritoneal cavity. Take a paraesophageal hernia—there is little opportunity for strangulation as in a femoral or inguinal hernia.

A pleuroperitoneal hernia most closely simulates an external hernia in its threat of strangulation. Yet, even here, we are dealing with muscular tissues and not with unyielding ligaments.

Harrington, of Rochester, a number of years ago suggested in diaphragmatic hernia crushing of the phrenic nerve. It might be of some value in relaxing the diaphragm in a pleuroperitoneal hernia.

Some of you may remember a paper of about 25 years ago by Truesdale, of Fall River, Massachusetts. He talked about bumper fractures of the diaphragm in children. If you and I crossed the street, and were hit by an oncoming automobile, we would probably have our tibias broken. A small child in the same circumstance would be struck in the belly and might have a ruptured liver, or the diaphragm might rupture and the stomach, colon, and small intestine would find their way up there. Truesdale suggested the use of a cecostomy, to relieve obstruction in these cases. Today we would get at the problem a little more directly, by use of inlying gastric suction.

Paraduodenal hernia is not a rare hernia. Obstruction, however, in these sacs is not a frequent occurrence. It runs in my mind that an English surgeon by the name of Taylor, in the *British Journal of Surgery* in 1930, described preoperative recognition of the presence of the gut in such a sac-like arrangement. Taylor stressed the appearance, especially on the lower side of a rounded sac-like appearance of the gut—occasioned by the inferior surface of the paraduodenal peritoneal sac.

I have encountered paraesophageal herniae not infrequently when operating for gastric cancer or for peptic ulcer. If on opening the peritoneal cavity there is little nor no free gut—the diagnosis of paraduodenal hernia comes immediately to mind.

Before completing the operation, one pulls the bowel out, and stitches the edges of the peritoneal sac together, thus doing away with the hernial orifice. The other thing one might do, of course, is to cut the sac widely open, observing carefully not to divide the vessels which run in the anterior margin of the sac. One can then tack the peritoneum to the posterior abdominal wall.

The foramen of Winslow hernia is a treacherous and an interesting one. I think some have been diagnosed preoperatively, largely by the location of the distended loops of gut and their fixation. I haven't personally had to wrestle with the problem of a strangulated hernia in the foramen of Winslow. The hernia into

the intersigmoid fossa—a little dimple in the mesentery under the left ureter, first described by J. B. Murphy, is a fairly common internal hernia, and a number of cases have now been reported and successfully treated. Strangulated pericecal hernia, in my experience, is also rare.

Now, as one considers the broader field mentioned by Dr. Moseley, of apertures in the mesocolon or mesentery, there are several conditions which come to mind. Dr. Snapper referred to iatrogenic herniae in which the surgeon creates the hernial orifice through an antecedant abdominal operation. They are common and very interesting. These projections of bowel into such apertures are probably the most common of all internal herniae.

A single fibrin strand left on a loop of gut can be the cause of much misery to the patient long after operation—a circumstance which may be very difficult to recognize. I am astounded myself occasionally as to how long it has taken to come to the understanding that the patient has an obstruction. One operates upon a patient for peptic ulcer doing a gastric resection. Years go by and he isn't quite relieved, and you are worried largely about the dumping syndrome. You listen to the story and see the patient. You observe he has intestinal colic, and finally conclude that the symptoms have been those of recurrent bowel obstruction. One needs to keep this possibility in mind in dealing with patients believed to be suffering from the dumping syndrome.

When one operates for any situation in the peritoneal cavity, it is wise to place one's hand down into the pelvis. I have encountered rather frequently long arches of epiploic tags, fused with each other, thus constituting a potential hernial orifice. I have seen this situation many times. I am always at pains to cut these arches for I feel they are a rather common cause of obstruction, especially in the aged. Moreover, to avoid leaving bits of fibrin in the peritoneal cavity, I have long followed the practice of washing out the peritoneal cavity with copious quantities of saline solution on the completion of long and difficult operations.

Dr. Snapper referred to an interesting circumstance: the possibility of diagnosing the escape of air in a strangulating obstruction. In the light of what was said, I think one must accept the explanation that the gas certainly was in the bowel, but, as Dr. Snapper indicated, one of the early signs of actual perforation, and a serious threat to the peritoneal cavity is the appearance of gas when you can see it outside the gut wall. You just see the interior of the gut wall when you are looking at gas in the intestine, but when you see the outer wall of the gut on the x-ray film, there is usually gas outside, and it is a pretty good sign of loss of viability of the gut.

Now, as I said yesterday in discussing Dr. Flood's paper on peptic ulcer, there has been a very gratifying improvement in mortality in appendicitis and bowel obstruction, but not at all in the mortality of peptic ulcer. It is interesting that there has been approximately a 50 per cent reduction in mortality of bowel obstruction. If one takes the figures of the Metropolitan Life Insurance Company,

which concerns largely policy-holders, between the ages of 15 and 65, it is agreed that the risk of bowel obstruction has been greatly improved, there being only about two deaths per hundred thousand for that age group. The large mortalities today in bowel obstruction concern two age groups—infants and the aged. The reason that the mortality of bowel obstruction continues high in infants is because of serious obstruction like meconium ileus, and congenital atresia—conditions which will always be very difficult to manage. Not so many years ago, there were no successes to report in meconium ileus or in intestinal atresia.

At the other extremity of life, age itself is, of course, an important item in the mortality. Yet too many surgeons have employed cecostomy to decompress a greatly distended colon when a transverse colostomy is simpler and far safer for acute colic obstruction due to cancer of the left colon.

How has the reduction in mortality in bowel obstruction come about? Through a number of things including recognition that obstruction is present. We also have better trained surgeons than we had 20 years ago. We have intubation; our knowledge of pre- and postoperative care is infinitely better than it was then. Our objective, of course, in the management of bowel obstruction should be to deal with the primary condition as though distention were not present. In many of the instances related by Drs. Nathan and Moseley, the obstructive agents were bands. How simple to cut a band when obstruction is not present, but how treacherous to deal with bands when one has to deal also with a sea of unruly gut such as is usually present in bowel obstruction.

Careful anesthesia and avoidance of regurgitation into the lungs are essential. At one time it was said by some that if one operated for bowel obstruction and used any other anesthetic than spinal, one should be tried before a jury of his peers. It was considered that serious by some. I employ only general anesthesia. I use an intratracheal tube, which protects against regurgitation into the lungs. Spillage is dangerous in the presence of bowel obstruction. And, of course, an inlying gastric tube has been employed to make certain that the stomach has been emptied and is kept empty during operation. One then gets at the business of dealing with the distended gut. Even in 1952, with many antibiotics available, it is still important to employ aseptic decompression of the gut at operation. If visible spillage occurs, the risk is pyramided despite having antibiotics. Ten years ago I made this assertion before an important surgical body. Upon conclusion of the discussion, several surgeons descended upon me and said, "You don't mean spillage; you must mean contamination." I retorted: "No, I mean spillage, visible spillage". Yes, even in 1952, surgeons should strive mightily to avoid spillage when decompressing the distended, obstructed gut. Spillage pyramids the risk. Why employ crude, worn-out methods to decompress the distended gut at operation, when an aseptic decompressive suction enterotomy can be done just as easily. I have said all of this in a monograph on bowel obstruction. There is, therefore, little need to belabor the point unduly here.

COMMENTS ON DISEASES OF THE SMALL INTESTINE*

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Although considerable progress has been made in the field of diseases of the small intestine in the past two decades, this important segment of the gastrointestinal tract still remains the stepchild of clinical gastroenterology. The fact that the discussion of the diseases of the small intestine was left for the last day of this course might thus be interpreted.

The Small Intestine—The Neglected Part of Gastroenterology

The attention of the clinician in his routine gastrointestinal work centers around the esophagus, the stomach and then, after a detour of many feet of small bowel, the colon and of course the liver, the biliary tree and the pancreas. The reason for the detour may be found in well taken statements in our textbooks, one of which reads as follows: "The small bowel, other than the duodenum, is rarely the site of pathologic processes, although the small bowel is one of the most important parts of the digestive tract".

It is true that the dreaded carcinoma of the gastrointestinal tract rarely affects the small bowel. Another important disease entity, peptic ulcer, may be localized in the lower esophagus, in the stomach or duodenum and only infrequently in the small bowel as an anastomotic jejunal ulcer or in a Meckel's diverticulum; but even in the latter unusual location, the ulcer originates in an island of aberrant gastric mucosa. In contrast to these comparatively rare disorders, the physiologic role of the small intestine in digestion, absorption and alimentation makes it the most important part of the gastrointestinal tract. Diseases of the small intestine have an extremely profound effect on the nutritional status of the individual. As a rule, obstruction of the small bowel results in more severe general symptoms than in that of the colon. With the exception of advanced malignancy, few other conditions produce more severe malnutrition, more loss of weight and cachexia, than disorders of the small intestine, e.g., long-standing sprue or extensive ileojejunitis. But these disorders do not compete in frequency with carcinoma or peptic ulcer.

There are still other reasons which explain why the small bowel has been greatly neglected in our thinking and study of the gastrointestinal tract. One reason is the anatomic location of the small intestine. It is preceded by the stomach and followed by the colon. Therefore, many symptoms and signs caused

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by disorders of the small intestine have been attributed to either the stomach or to the colon. One may reason for instance that in acute chronic gastritis an extension of the inflammatory process into the upper small bowel would occur not infrequently, although such involvement would be difficult to establish. The diagnosis of disorders of the small bowel is made often by inference and by exclusion of gastric and/or colonic pathology.

Diagnostic Procedures

Another difficulty may be seen in the elaboration of newer diagnostic methods in gastroenterology which again are applicable to a greater extent to diseases of the esophagus, stomach and colon than to those of the small intestine. We are referring to esophagoscopy, gastroscopy, proctosigmoidoscopy, the newer methods of intubation and the refined radioscopic and radiographic technics.

In diseases of the small intestine *physical diagnosis* may be of great help. Abnormal peristaltic waves may be seen in obstruction of the small intestine and the contours of a large tumor may be visible. One may be able to palpate a tumor or a large granulomatous mass. Auscultation may aid in differentiating atonic and spastic ileus. Deep tenderness around the mid-abdomen to the right and left of the umbilicus may reflect on pathological states of the small bowel. Zones of hyperesthesia located at the same level have been also related to the small intestine ("Duenndarmdruckpunkt").

Direct visualization used in diseases of the esophagus, stomach and lower colon cannot be applied to the small intestine except, rarely, when the jejunum is anastomosed directly to the stomach and a small portion of it becomes visible to the gastroscope. In ileostomy the adjacent parts of the ileum may be observed by direct inspection.

The procedure of obtaining secretions of the small intestine for study is circumstantial and time-consuming; it is important to separate the intestinal secretions from those of the stomach, the liver and the pancreas. Thus, the diagnostic information obtained by the use of the intubation technics is of limited value in diseases of the small intestine; additional studies on intestinal secretions and especially on the composition of the *succus entericus* would be of great value.

Considerable advances have been made recently in the *x-ray diagnosis* of diseases of the small intestine. In many instances roentgen observation represents the only objective diagnostic basis. We are somewhat skeptical about the value of an "intermediary film" taken in the course of a roentgen study of the gastrointestinal tract 2 to 3 hours after ingestion of barium and we do not believe that it can replace a small intestine series. It may perhaps occasionally call attention to an existing abnormality of the small bowel; in other instances and especially in those associated with hypermotility a pathologic lesion may be completely missed.

A small intestine roentgen study requires ingestion of a small or medium quantity of barium. With large quantities of barium overfilling and overlapping of the loops is produced. The study must include both fluoroscopic observation and radiography at frequent intervals (half to one hour) until the bulk of the barium appears in the colon.

Because of great variations in the appearance of the small bowel, the roentgen study must be repeated often. Lesions suspicious of granulomatous ileitis may, on re-examination, prove to be caused by the spasm of an acute enteritis or by allergy or may be the result of a drastic laxative. As a matter of fact, we are lacking in knowledge of the effects of irritants, such as mustard and other spices, digitalis and other drugs, upon the small intestine. By analogy one can only suspect that over-indulgence in certain irritating foods, alcohol and certain drugs may be the cause of acute gastric as well as small intestinal irritation (acute gastritis, acute enteritis).

Fecal analysis may be an important source of information in the study of the small bowel. It is frequently neglected or inadequately done. The customary important search for parasites, ova and pathogenic organisms represents the first step only. Mucus in small particles well mixed with the feces may have its origin in the small intestine or in the proximal colon in contrast to the large pieces or bands of mucus which originate in the distal part of the large bowel. Green discoloration of the mucus of the stool by biliverdin again incriminates the small bowel and indicates a very rapid transit through the small bowel ("jejunal diarrhea").

The microscopic study of a simple smear preparation of the stool may offer valuable information concerning undigested fat which may be seen as neutral fat, fatty acids and fatty soaps; large quantities of fat may indicate defective fat absorption (steatorrhea). (Chemical determination of fecal fat permits quantitative estimation of steatorrhea). Muscle fibres may be easily detected by their striation and, if present in large numbers, may indicate deficient digestion of meat (creatorrhea). By the addition of a drop of Lugol's solution undigested starch cells may be identified by their color. The simple determination of the chemical reaction of the stool with litmus paper may offer important information; e.g. acid reaction will be in accord with the finding of large quantities of non-absorbed starch causing increased intestinal fermentation.

It is of course important to place the patient on a special minimum-load type of diet prior to these fecal studies. This need not necessarily be the original Schmidt test diet, but the principle of avoiding large quantities of roughage and of administering a well balanced ration of easily absorbable protein, carbohydrate and fat including an adequate supply of vitamins and minerals must be observed.

The elaboration of various *absorption tests* is important for the study of the physiology and the pathology of the small bowel. For practical purposes an

oral glucose tolerance test is a good index of absorption of monosaccharides. If its course is found to be flat by the oral route and normal in shape by the intravenous route, then disturbed intestinal absorption may be the cause of this difference. The oral galactose tolerance test has been similarly used[1]. Oral tests with standard doses of glycine have been employed to measure the capacity of the intestine to absorb amino acids[2]. Fat and Vitamin A tolerance tests are used as indices of digestion and absorption of fats and fat soluble vitamins[3]. They are also valuable indices of small intestinal absorption after partial and total gastrectomy and after small intestinal resection[4]. For additional information on absorption tests the reader may refer to the recently published summary of Janowitz[5].

Extensive Resection of the Small Intestine

Following resection of large segments of the small bowel, grave nutritional difficulties may arise. Absorption tests discussed above, as well as metabolic balance studies may be used in order to evaluate the resulting defect of absorption. It is of interest that 4 to 5 feet of jejunum anastomosed to the colon are compatible with life although strict diet regulation, administration of alimentary supplements, vitamins, liver extract and Vitamin B_{12} may be necessary[4]. More extensive resections of the small bowel performed for wide spread disease or gangrene may produce insurmountable nutritional difficulties resulting in severely negative nitrogen, fat and electrolyte balances and may lead to fatal termination.

Tumors of the Small Intestine

Tumors of the small intestine are comparatively rare. It is as if nature wished to show great respect for the vital functions of the small bowel and wanted to make this part of the gastrointestinal tract in some way "immune" to carcinoma. Sarcoma and lymphosarcoma are also rare occurrences. Benign tumors of the small bowel, fibroma, leiomyoma, angioma, cysts, etc., are being diagnosed with increasing frequency and are being subjected to surgical therapy.

The Sprue Syndrome

The absorptive disorders of the small bowel represent an interesting chapter of gastroenterology. They are characterized by diarrhea, poor absorption of food, especially fats, and absence of inflammatory disease of the small bowel. Enormous degrees of dilatation and atrophy of the gut may be encountered at autopsy. These conditions are usually considered under the terms tropical or nontropical sprue, idiopathic steatorrhea or celiac disease. At present the opinion prevails that these disorders represent various manifestations of the sprue syndrome. The etiology of primary sprue is obscure; it usually commences as an extensive nutritional deficiency. Recent observations indicate that the sprue syndrome represents a genetically transmitted error of metabolism. The intestinal symptoms and signs may be then interpreted as a manifestation of a more

profound systemic disorder[4], precipitated by various environmental factors, e.g., nutritional deficiency and infection.

The sprue syndrome is more frequent than was originally believed. It is certainly present in the population of our large cities in persons who were born and had lived exclusively in non-tropical climates. Whenever there is a history of considerable weight loss, sore mouth, anemia (and not necessarily a hyperchromic macrocytic anemia) and diarrhea, sprue is a diagnostic possibility. If the blood picture is indicative of pernicious anemia which is associated with voluminous light colored stools the diagnosis of sprue will often replace that of pernicious anemia. Additional symptoms and signs in the severe variety of sprue may be hypocalcemia and tetany, osteoporosis, hypoproteinemia and various forms of vitamin deficiency.

It is advisable to separate primary idiopathic sprue from secondary sprue[6]. Primary sprue is a syndrome in which no roentgenologic or other evidences of gross organic disease can account for the signs and symptoms. The term secondary sprue should be reserved for patients with generalized disease of the small intestine responsible for the disorder. Lipodystrophia intestinalis (Whipple's disease), extensive lymphosarcoma or amyloidosis of the small intestine are examples of gross pathological disorders causing secondary sprue.

Roentgen study of the gastrointestinal tract in primary sprue usually reveals an abnormal pattern of the small bowel characterized by enormous dilatation of jejunal segments, increased secretion, scattering of the barium and unusual type of segmentation. While each of these features may occur in a wide variety of disorders such as nephrosis, hyperthyroidism, pancreatic steatorrhea, pellagra and other deficiency states, the combination of all these symptoms, the typical localization and intensity are more frequent in primary sprue[7]. The differential diagnosis of the sprue syndrome must of course include organic diseases of the small bowel such as lymphosarcoma, ileojejunitis and tuberculosis[7].

Standard Sprue Therapy

The treatment of primary sprue is based on empirical observation and consists essentially of a high protein diet with limited amounts of fats and starches[8]; certain monosaccharides (fructose) are strikingly well tolerated and may explain the wide use of bananas, strawberries and mangos in sprue. Adequate quantities of liver extract, Vitamin B_{12} and/or folic acid should supplement the sprue diet. The response to standard therapy varies with the individual. In the tropical variety of sprue there are many instances of complete cures without relapse[9]. The majority of patients require continuous therapy, particularly those with the non-tropical form of sprue. Even under these circumstances complete remission often cannot be maintained so that despite normal hematological status diarrhea and steatorrhea, underweight, weakness and signs of avitaminosis are present. There is a group of patients who after many years of satisfactory response to

standard sprue therapy become resistant and others who prove refractory to most intensive courses of treatment even in the early phases of sprue.

Management of Intractable Sprue

With cases of refractory sprue the physician feels the need for additional therapeutic possibilities. The debatable role of the adrenal cortex in intestinal absorption suggested the use of adrenal cortical therapy. A considerable group of our patients have been under this form of therapy for over two years[10,11,12]. Corticotropin, used parenterally, cortisone acetate and hydrocortisone free alcohol used orally do not represent curative therapy in sprue; however, a clinical remission, the extent of which varied, was observed in all patients. The important clinical changes were subsidence of diarrhea, increased sense of well-being, improved and often ravenous appetite, and considerable gain in body weight. In some instances there was marked increase in the serum albumin and the serum calcium. There was evidence that administration of these hormones in sprue improves intestinal absorption of fat, and fat soluble vitamins. The lower fecal fat content, the higher Vitamin A and carotene levels of the serum and occasionally, improved Vitamin A tolerance tests are indicative of such a change. Nevertheless one should not overlook the possibilty that the effect of these drugs on appetite and mood might have contributed to the observed results.

Multiple courses of treatment with these hormones resulted in clinical improvement in each case and no resistance to this form of therapy has so far developed. One to six weeks after cessation of hormonal therapy, however, clinical relapse invariably occurred. The duration of remission without hormonal therapy depended upon the dosage previously used. In patients who had been maintained with minimal doses just sufficient to suppress the clinical symptoms, discontinuation of the therapy resulted in complete relapse within a few days; in contrast, patients who had been maintained with larger doses of the drug remained in remission for a few weeks. Our policy has therefore been to maintain patients with refractory sprue on continuous therapy with minimal effective doses of cortisone, hydrocortisone free alcohol or corticotropin[11,12]. It might be mentioned parenthetically that hydrocortisone acetate proved to be poorly absorbed and therefore ineffective. For the other hormones the minimal maintenance dose was established for each patient by prolonged "clinical titration". Recent studies seem to indicate that hydrocortisone free alcohol offered definite advantages in comparison with cortisone. It is effective in smaller doses without producing undesired side-effects[12]. Similar observations have been made in the use of this steroid in rheumatoid arthritis[13].

Although the treatment of refractory sprue with adrenocortical therapy is not curative it represents a great advance in the management of patients with this disorder. We have observed patients who, because of their malnutrition, weakness and diarrhea, were condemned to complete or semi-invalidism and who,

under adrenocortical therapy, became able to lead a normal life and to work normally.

One may reason perhaps that neither cortisone nor hydrocortisone are primarily involved in the etiology of sprue and that other related compounds or analogues may in the future prove even more effective in the management of this disorder.

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Discussion

Question:—I am interested to know whether there has been any work on cortisone and ACTH in tropical sprue.

Dr. Adlersberg:—Dr. Milanes, of Havana, Cuba, said in a discussion of my paper that he had treated a few cases of tropical sprue, resistant to ordinary therapy, with cortisone, and has observed good results.

Dr. I. Snapper:—It is well-known that under influence of the function of the adrenals, phosphorylation of glucose takes place in the intestine which facilitates the absorption of glucose. This may be one of the reasons why ACTH and cortisone may also improve the absorption of other substances.

Dr. Shecket:—Is there remission or change in the x-ray picture?

Dr. Snapper:—Was there improvement in the x-ray picture after ACTH or cortisone?

Dr. Adlersberg:—It varies with the patient. Only rarely does one find in these patients definite improvement of the roentgen pattern of the small intestine. As a rule there is a discrepancy between the remarkable clinical improvement and persistence of the roentgen changes of the small intestine. This discrepancy might indicate that the improvement observed in sprue is only symptomatic.

The same is true in regard to fecal fat. In some of these patients we saw marked reduction of steatorrhea. Others did very well, although the fat content of the formed feces continued to be high; one must consider, however, that parallel with the clinical improvement, the patients' fat consumption was considerably raised (from 30 gm. to 100 gm. daily). The most striking thing is that those who had hypoproteinemia were able to fill up their blood proteins and to rebuild their muscles.

Dr. Snapper:—Formerly the designation sprue was reserved for a disease which occurred in the tropics, only in white people, never in Chinese, never in Indonesians. Such sprue patients developed fatty diarrhea, anemia, painful red tongue and abdominal distention. At autopsy, nothing was found. This tropical sprue was a dangerous disease which was often fatal. Remarkably enough, a fruit diet, especially a strawberry diet, improved the condition temporarily, but removal to a cool climate was usually necessary. When liver treatment was introduced, the outlook changed completely, because under the influence of liver injections, the condition of these sprue patients improved immediately.

This tropical sprue was due to poor absorption in the small intestine. Gradually it has become customary to designate all fatty diarrheas due to poor intestinal absorption as sprue, irrespective whether they occur in the tropics or in temperate zones. Nevertheless non-tropical sprue may well be a different disease from that which is so frequent in the tropics, even if in both diseases fatty diarrhea due to poor intestinal absorption is the outstanding sign.

So-called non-tropical sprue can usually be distinguished from tropical sprue because:—

a. the disease develops in a cool climate

b. it frequently does not react upon liver treatment

c. usually clubbing of fingers develop, whereas clubbing of fingers does not occur in tropical sprue.

Tropical and non-tropical sprue have, however, one important fact in common, the etiology of the faulty intestinal absorption is not known.

How can we differentiate these sprue-like syndromes from other diseases with fatty diarrhea? In pancreatic disease, fatty diarrhea is present because in the absence of the lipase of the pancreatic juice, no hydrolysis of fat takes place in the intestine.

The differential diagnosis can usually be made without difficulties. Since in sprue-like conditions, the intestinal absorption is poor, glucose given by mouth does not give rise to the normally occurring hyperglycemia and the blood sugar curve remains flat. The same holds true when instead of glucose, Vitamin A is administered as has been so clearly demonstrated by Dr. Adlersberg and his co-workers. In contrast the blood sugar curve is usually diabetic in character when the fatty diarrhea is due to impaired function of the pancreas. The blood sugar curves obtained after pancreatic removal are the same as found in pancreatic deficiency.

Since in diseases of the pancreas, fatty diarrhea is due to the absence of lipase which leads to insufficient hydrolysis of neutral fats, one might expect that in pancreatic disease the stool will contain only neutral fat. This actually occurs in many cases of pancreatic diseases. In other cases of pancreatic insufficiency, however, the neutral fat which because of the absence of lipase is not hydrolyzed in the small intestine, is hydrolyzed by bacteria after the nonabsorbed fat has reached the colon. Since fatty acids are only absorbed in the small intestine, the fatty acids formed in the colon appear in the stool. Thus there are cases of pancreatic disease where due to bacterial influence, the fats are eliminated in the stool in the form of fatty acids.

It has already been mentioned that the treatment of non-tropical sprue with liver, folic acid and a sprue diet has in most cases given unsatisfactory results. One can only agree with Dr. Adlersberg that many of these persistent cases do react upon treatment with ACTH or cortisone. Thanks to these drugs, the lives of many of these patients with non-tropical sprue has become much more tolerable.

PHYSIOPATHOLOGICAL CHANGES IN UPPER GASTROINTESTINAL BLEEDING*

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Upper gastrointestinal bleeding presents a diagnostic and therapeutic problem which has been extensively discussed in the literature. Relatively few reviews have appeared on the physiological changes in the patient upon which the diagnosis and treatment must rest. This paper is a review of the literature on this physiology with illustrations from recent cases in this hospital.

The patient with upper gastrointestinal bleeding has been described as presenting anemia, depleted blood volume, starvation, dehydration, uremia, acidosis, chloride and protein deficiencies. The patient may also have fever, leucocytosis, and changes in the liver, heart, and central nervous system[1].

The incidence of fever varies with its definition. Bang[2] reported 91 per cent of his patients over 37° C. and Purjesz[3], 73 per cent. Dill and Isenhour[4] reported 44 cases with 80 per cent having a temperature over 99.2° F. once, or over 99° F. for two days. Forty-six of his patients with nonbleeding ulcers and 37 per cent of patients described as neurotic were febrile by the same standards. Selesnick and White[5] in reporting 100 cases found only 47 per cent with an oral temperature of over 99.1° F. if not transfused, but with 85 per cent of transfused patients over 99.1° F. There was a direct correlation between the number of transfusions and the fever incidence. Experimentally bleeding dogs of 25 per cent of their blood volume and refeeding the blood produced no significant rise in temperature nor did feeding humans up to 1,800 c.c. of citrated blood[4,7].

The respiratory rate is not consistently elevated[6,55]. The B.M.R. has been determined in bleeding patients and found to be normal[9].

With the onset of bleeding the blood pressure rises slightly and then decreases as the blood volume and the cardiac output fall[12,51]. With a decrease in blood volume the pulse rate is increased. This has been the basis for a test for blood volume loss[10]. After the blood pressure and pulse stabilize, the patient is tilted to 75° on a tilt table. The pulse rate will accelerate roughly proportionally to the blood loss, i.e., the rate will increase 20 beats per minute with a loss of 5-9 c.c. of blood per kilogram body weight, and 40 beats for 0-14 c.c. With a greater loss the blood pressure falls, as does the pulse after the initial rise.

The hemoglobin follows the usual pattern in hemorrhage with a fall delayed until the plasma volume has increased to replace the blood volume loss. The

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hemoglobin fall is proportional to the blood volume loss. The time factors have been estimated by Bennett et al[11]. Leucocytosis has been described up to 20,000. Dill and Isenhour[4] found an average count of 8,000 in both bleeding and non-bleeding ulcer patients. Citrated blood has been fed to humans in doses up to 1,800 c.c. without producing leucocytosis[7]. The ESR has been reported as normal[8].

Moss and Schiff[13] reported normal platelet counts, plasma fibrinogen levels, prothrombin time, and clotting time in their patients. The bleeding time was normal. If a pressure of 40 mm. of mercury was applied above the puncture point, about ¼ of patients showed prolonged bleeding time. The significance of this test is unknown. No correlation was found between the plasma Vitamin C levels, the capillary fragility, and the incidence of gastrointestinal bleeding in a large clinic series[14]. Calcium levels also were not related to bleeding in these patients[13].

In 1934 Sanguinetti reported an elevated blood urea in patients with bleeding peptic ulcers[15]. This observation was extended by many case reports[1,16-20,28] and by attempts to duplicate this rise experimentally. The highest figures found in patients without renal disease were 145 mg. per cent[15], 140 mg. per cent[17] and 105 mg. per cent in three patients respectively[20].

In 1942 Black[7] reviewed several series and noted that:

1. Seventy-five per cent of patients admitted to hospital showed a blood urea over 50 mg. per cent. (This is equivalent to a BUN of 23.3 mg. per cent).

2. The BUN rise might occur as soon as two hours after the onset of clinical bleeding, and the peak figure was ordinarily reached within the first two days after the initial bleeding.

3. Urinary output tended to fall during the first 48 hours and then to rise to diuresia.

4. Urea clearance tended to fall while the BUN was rising.

5. Patients remained in positive nitrogen balance.

6. Urine chloride excretion was generally low.

7. Plasma creatinine, uric acid, chlorides, bicarbonate, and total base were within normal limits or were inconsistently variable.

Since the BUN is the resultant of the rate of urea formation and the rate of urea excretion, this scheme was proposed[7]:

Sources of the urea:—

1. Nitrogen absorbed from the breakdown of the ingested blood.

2. Protein breakdown in body tissues to amino acids to be deaminated by the liver.

3. Abnormal liver activity with an increased urea production.

Factors affecting excretion:—

1. Previous latent renal lesions which are only revealed by the increased demands on the kidney to excrete urea.

2. Fall in blood pressure with decreased renal blood flow.

3. Fluid and electrolyte imbalance with impaired renal function.

Chunn and Harkins[21-24] produced a BUN rise of 15-25 mg. per cent in 5-10 hours following intragastric administration of blood in the healthy dog, and found that the rise in BUN was proportional to the amount of red cells fed, and that the rise occurred only with blood released in the upper gastrointestinal tract. Kaump and Parsons[25] found that in animals both bled and fed citrate blood, a diphasic curve was obtained, with a BUN rise of 25-35 mg. per cent, in the first 24 hours after feeding with 250-1,000 c.c. of blood and a second rise of 2-25 mg. per cent at 48 hours attributed to anemia.

The most extensive feeding experiments were those of Gregory[26]. He simultaneously bled and fed dogs their own citrated blood by stomach tube. Feeding produced a rise to 25-30 mg. per cent and if the dogs were bled to a systolic pressure of 80 mm., the BUN would go to 25-40 mg. per cent. There was no uremia with just anemia. In a general way a low urea clearance was associated with an elevated BUN. Well hydrated animals had a lower BUN regardless of clearance. Schiff et al[27] noted that the BUN rose most rapidly with a single large bleeding episode and was largest with intragastric hemorrhages.

Greenblatt and Cohn[29] made a quantitative determination of the blood loss in humans necessary to produce a BUN elevation. Five volunteers were bled of 500 c.c. and the blood reingested by stomach tube. The BUN rose 24 per cent above fasting levels in 24 hours, but not above normal values. When three volunteers were bled of 800 c.c. and the blood reingested, their BUN rose to 90 per cent above fasting levels, while at the same time their urea clearance fell to 65 per cent of normal.

Because the BUN may rise within 2 hours after the onset of bleeding, while the BUN in feeding experiment does not rise for 12-36 hours, body protein breakdown has been suggested as a nitrogen source[7]. Balance experiments are difficult as only about 80 gm. of protein would be required to furnish a BUN rise of 25 mg. per cent. Black's patients lost weight even if well hydrated, which in general they were not on admission[30]. The catabolism of body protein in stress situations is well documented[31].

Altered liver function has been suggested as a source for the urea. When the liver blood flow is reduced the BUN generally falls, with a rise in amino acid blood levels[32,33].

A report on renal clearance in gastrointestinal bleeders was published by Borst[34] who determined urea clearances, as well as serum sodium, potassium, and chloride levels in patients who were approaching shock. His patients had all had high oral fluid intakes prior to the beginning of the study. He found hyperchloremia and azotemia. The urine contained small amounts of sodium chloride. The urea clearance decreased while the patient was in shock. Sodium excretion began when the patient had recovered from shock, when the hemoglobin was at its lowest, the BUN down, and urinary urea falling. Potassium excretion remained high throughout with normal to low serum potassium levels. Because of the high potassium excretion Borst raised the question of the role of the adrenal gland in the body's response to gastrointestinal hemorrhage. Selye[35] noted this work as a typical example of stress reaction.

Johnson[36], in 17 patients, noted a BUN of over 25 mg. per cent only if the urea clearance was decreased to at last 50 per cent. When blood was fed to posthemorrhagic patients he obtained a rise to 25 mg. per cent, and in a patient with a urea clearance of 50 per cent, a rise to 50 mg. per cent. He found the urea clearance did not return to normal for four weeks. Black, Powell and Smith[37], with three severe bleeders, found that the inulin clearance might be normal or depressed with an elevated BUN, and was normal to high during recovery. Diodrast clearances were depressed relatively more than the inulin. They attributed these effects to decreased plasma flow. They noted no relation between renal blood flow and blood pressure or blood volume. Stevens and Schiff[38] found that of four cases with elevated BUN, two had normal urea, inulin and phenol red clearances. Previous renal diseases have also been suggested as an etiology for the elevated BUN[39] but the high incidence of elevated BUN with gastrointestinal bleeding makes this seem an occasional contributing factor at most.

The consensus among the quoted authors is that a combination of absorbed blood, body protein breakdown, and minimally to moderately diminished renal function accounts for the elevated BUN, with one factor or another more significant in the individual case. Since the BUN is proportional to the amount of blood in the gastrointestinal tract[22] and tends to decrease after the second day if bleeding does not continue[7], the daily BUN level has been suggested as an aid in prognosis[20,49]. Reports on both sides of this question have appeared[40,41].

The normal liver is only slightly affected by the usual gastrointestinal hemorrhage. Navarro et al[42] reported nine cases in which an elevated serum bilirubin was found only in those cases proven to have liver disease by biopsy or postmortem. Zamcheck et al[43] used the BSP test as a differential between esophageal varices and peptic ulcer. They divided 61 cases of bleeding into three groups: I. advanced liver disease, II. other liver disease, III. no liver disease. In group I after hemorrhage the BSP retention varied from 17-60 per cent, in group II from 3-33 per cent, and Group III from 0-17 per cent. In group III those patients in severe shock showed values from 8-17 per cent, while of those in mild shock four out of fourteen had values from 6-13 per cent. Of those not in shock only

three out of thirteen showed values from 6-13 per cent. Most of the abnormal values were in patients over 60, and all returned to normal in five days, except in Group I. These results correspond to experiments showing reduced BSP clearance and by inference reduced hepatic blood flow in bled dogs[44,45].

Case 1:—C. H., a 25 year old white male, was admitted 10/1/52 with the complaint of hematemesis for 12 hours. Patient had had no previous digestive disease, ate well but occasionally drank a glass of beer. On the morning of admission he had noticed sharp epigastric pain and vomited up about a quart of bloody material. Physical findings on admission showed a B.P. 120/90, a pulse of 90, respirations of 20, mild pallor, distant heart sounds, mild epigastric tenderness, and clammy extremities. Admission laboratory work: Hgb. 16.2 gms., WBC 12,000, BUN 19.9 mg. per cent. The patient passed bloody stool and vomitus the first day estimated at 2,000 c.c. On 10/2 following multiple transfusions the Hgb. was 10.4 gm. the BSP 44 per cent. The patient continued to bleed and on 10/3 he was explored surgically and a bleeding duodenal ulcer demonstrated which was treated by subtotal resection of the stomach. The patient did well postoperatively and on 10/17 the Bilirubin was 0.2 mg. per cent, BSP 10.5 per cent, alk. phosphatase 2.5, Bodansky units, cephalin flocculation neg., thymol turbidity 3.5 and the A/G ratio 4.7/2.6.

The motility of the gastrointestinal tract may be affected by bleeding. Hesser[46] found that a carbon meal passed through the normal tract in 24 hours. When patients were given a barium sulfate swallow on the day of bleeding their stools became negative for barium only in 8-21 days, and became guaic negative about three days after they became negative for barium.

The central nervous system may be affected by gastrointestinal bleeding. Lazege[47] reported generalized convulsions which subsided with transfusions. Ophthalmologists[48,49] have repeatedly reported blindness following hemorrhage, with optic nerve atrophy and central scotomata. These changes may persist and may be accompanied by permanent mental changes. The ophthalmoscopic findings associated with the atrophy appear on the first to third day after hemorrhage.

Case 2:—E. M., a 65 year old white salesman was admitted on 2/13/53 with a history of tarry stools for three days and hematemesis the evening before and the afternoon just prior to admission, with total loss estimated as over two quarts. He had had mild indigestion for years and bloating the past 5-6 weeks. G.I. series in the past had been negative. Physical examination showed a sallow white male, weak and apprehensive, and passing bloody stool during the examination. B.P. 60/20, pulse 130, respirations 24, temperature 100.2°. The skin was clammy. There was no abdominal tenderness. Hgb. on 2/13 was 4.0 gm. Patient received multiple transfusions and came out of shock but continued to bleed rectally. On 2/14 Hgb. was 10.0 gm., WBC 10,500. Patient became irrational with constant masturbation and many fine tremors of hands, lips, and tongue, but no evidence of tetany. On 2/15 when the patient had apparently stopped bleeding the Hgb.

was 7.0 and the CO_2 59 vol. per cent. His temperature had risen to 103° and rose progressively to 105°. On 2/16 the BUN was 50 mg. per cent, the CO_2 73 vol. per cent, the sodium 139 meq., and the potassium 3.6 meq. The patient during his entire course had an excellent urinary output and received adequate fluids as well as blood. On 2/17 the patient's temperature rose to 107° without any change in his findings of disorientation, and twitching. He never showed convulsions or local weaknesses. On 2/17 he expired. Autopsy showed terminal pulmonary edema, and a duodenal ulcer with a small artery in the crater with the entire intestinal tract filled with blood. The brain showed no changes.

Case 3:—A. C., a 41 year old white housewife was admitted on 10/26/52 with the complaint of fainting and vomiting for three days. Five days prior to admission patient complained of a pounding frontal headache somewhat relieved by aspirin. One day later she noted spots in front of her eyes. During the three days prior to admission she fainted repeatedly, was dizzy and complained of persistent headaches. She also recalled that she had some blurring of vision and numbness of the left hand for one week. Irregular vomiting, non-bloody, had occurred during the five days prior to admission. Physical examination showed a well nourished female appearing pale and drowsy. B.P. was 120/70, pulse 120, respirations 20 and temperature 100.2°. The fundi were very pale and it was difficult to focus on discs or on vessels. The examination was otherwise negative. On 10/27 the hemoglobin was found to be 5.2 gm. The patient on 10/27 passed a bloody stool and had a frank hematemesis; at this time she admitted that she had melena at home. The patient received 3,500 c.c. of whole blood but continued to bleed. She was operated upon but no bleeding point was demonstrated. She ran a very stormy course postoperatively with a wound infection and phlebitis, but no important further bleeding. Pre- and postoperative G.I. films did not demonstrate a lesion. The patient's visual difficulties subsided postoperatively, and she had no further nervous symptoms except for an attack of migraine two weeks postoperatively.

Perhaps the most dramatic effect of upper gastrointestinal bleeding on other systems is the cardiovascular system. Rasmussen[50] reported E.K.G. changes in 21 out of 45 of his patients. Seventeen of these tracings were definitely abnormal, while 4 represented changes from a control. ST segments were depressed in leads II and III, and T waves inverted in II and III. These changes occurred early and reverted to normal with hemodilution. BUN changes roughly paralleled E.K.G. changes although not all patients with EKG changes had BUN rises. In one patient the same changes were produced by experimental anoxia several months later. Age distribution was the same for patients with and without changes, one patient of 17, having a positive tracing.

Master et al[51] reported coronary insufficiency in 59 of 103 cases. Six had angina without EKG changes, 34 had EKG changes without angina, and 19 had both. Twenty-two of 103 patients died with 18 showing evidence of coronary insufficiency. On autopsy, 15 showed subendocardial necrosis. The combination

of decreased coronary blood flow (despite lowered coronary resistance)[52] the tachycardia associated with the diminished output and the anemia secondary to hemorrhage was felt to be the cause, with the anemia a minor factor.

Anginal syndrome and myocardial infarction in patients with gastrointestinal bleeding have been reported in many additional papers[53]. Three of our patients primarily presented cardiac complaints, and in one bleeding was not discovered antemortem.

Case 4:—J. S., a 64 year old white male, was admitted 11/3/52 with the complaint of precordial pain seven hours in duration. The patient was awakened by pain which was pressing in character, associated with dizziness, nausea, profuse sweating and dyspnea. The patient had noted a chronic cough for three months, also a two-flight dyspnea, but had never had any chest pain, and denied any digestive symptoms. The patient had had a gallbladder operation, nature unknown, one year previously. Physical examination revealed a well-nourished elderly male appearing acutely ill. B.P. was 130/90, pulse 64, respirations 25, temperature 98.4°. Rales were heard at both bases. Heart sounds were distant. There was right upper quadrant tenderness. Admission Hgb. was 15.0 gm. and WBC 15,000. E.K.G. was interpreted as posterior wall necrosis. On the evening of admission the patient vomited up 500 c.c. of blood and his blood pressure fell to 90/72. On 11/4 he had further vomiting of blood and the pressure fell to 70/60 despite transfusions. On 11/4 the Hgb. was 14.0 gm., the BUN 40 mg. per cent. The patient stabilized after a total of 2,500 c.c. of blood. On 11/10 BUN was 10 mg. per cent. Patient had persistent dyspnea treated by aminophylline. G.I. series was negative. The patient's E.K.G. showed the usual progressive changes. The patient signed out after four weeks of hospitalization.

Case 5:—R. H., a 68 year old white actor, was admitted 3/24/53 with the complaint of shortness of breath for one week. Patient had had a myocardial infarction seven years ago, and seven months previously had been treated at our hospital for congestive heart failure and digitalized. One week prior to admission he had substernal pain lasting 12 hours, and since that time had had progressive dyspnea, orthopnea, paroxysmal nocturnal dyspnea, ankle edema, and has had intermittent chest pain. He had anorexia and nausea for the week. He had one episode of non-bloody vomiting.

Physical examination showed a well nourished elderly male acutely dyspneic, orthopneic and cyanotic. His neck veins were distended. His chest showed rales at both bases. The heart was enlarged, with poor sounds, and an irregular rhythm. The liver was down two fingers, and there was two plus ankle edema. B.P. was 94/60, the pulse 68, respirations 24 and temperature 100°. Admission laboratory results: Hgb. 15.4, WBC 14,000. BUN 52.1 mg. per cent. The patient improved somewhat in oxygen and with the usual adjuvants, but continued to have posterior rales in the chest and remained weak and anorexic. EKG showed shifting bundle branch block. The BUN continued to rise, to 111 on 3/30 and to

156 on 4/1 in the presence of a fair fluid intake and urine output and a specific gravity as high as 1.020 on casual urine specimens. His BUN on the previous admission had been normal. The patient expired on 4/3/53. An autopsy showed fluid in the right pleural cavity, pulmonary edema, enlarged left ventricle with an old anterior and revent posterior wall infarction. In addition the patient had blood throughout his intestines with three well marked duodenal ulcers, one of which contained a blood vessel. These appeared to be more than superficial. The kidneys showed some mild glomerular thickening.

This case particularly emphasizes the point of Chalmers et al[34] who reported on 101 cases of gastrointestinal fatal bleeding, in 25 per cent of whom the diagnosis of hemorrhage had not been made antemortem. A high degree of suspicion, particularly with a rising BUN and continued weakness and pallor might prove life-saving.

Summary

The literature has been reviewed with respect to vital signs, biochemical abnormal findings, and changes in the liver, heart, and central nervous system in patients with gastrointestinal bleeding. Case reports have been given to illustrate these points. It is pointed out that: 1. effective therapy depends on a knowledge of these factors, and 2. many cases of gastrointestinal bleeding may be masked by cardiac and central nervous system symptoms.

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THE EFFECTS OF PNEUMOPERITONEUM UPON THE DIGESTIVE TRACT

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Pneumoperitoneum, as a factor of therapeusis like other important medical achievements passed through the process of genesis, evolution, and final crystallization into a practical application. The subject is replete with the elements of accidental discovery, trial and error, and world-wide experimental effort by numerous able and gifted clinicians.

The application of pneumoperitoneum in the treatment of tuberculosis began in the early thirties. The treatment of tubercular peritonitis by laparotomy dates from 1872 by Spencer Wells. From then on, this procedure was frequently performed in the modified technics, by many investigators here and abroad, employing oxygen, oxygen and nitrogen, nitrogen alone, and finally air.

The gratifying results of this remedy created enthusiasm as indicated by its increasingly frequent use. At present, it has almost completely supplanted pneumothorax. A Dallas phthisiologist asked the following question: "What is pneumothorax anyway?" Briefly, experimentation has been done in peritoneal conditions: Tubercular peritonitis, in prophylaxis of peritoneal infection following abdominal surgery, septic peritonitis, and in the prevention of postoperative peritoneal adhesions. It was also applied in tuberculosis of the pelvic organs and rupture of a pyosalpinx, in pulmonary hemorrhage, occasionally in gastric bleeding due to ulcer, and esophageal varices with good results. In ascites due to liver cirrhosis it decreased the frequency of tapping. In certain cases of empyema, it restored the normal diaphragmatic excursions, thereby decreasing dyspnea and cyanosis. Fair results were indicated in tuberculous enteritis, intestinal ulceration and ulcerative colitis.

The theories expounded in favor of pneumoperitoneum are numerous. It is suggested that it increases the blood supply, produces immune bodies, creates local leucocytosis and venous hyperemia. The red cells are augmented after a primary leukopenia, followed by a leucocytosis, a high level of polynucleosis and eosinophylia. The oxygen has a direct bactericidal, bacteristatic and toxin neutralizing action. There is a mechanical effect exerting pressure on the vagi and sympathetic nerve endings, acting as an insulating layer between the intestinal loops, thereby preventing friction. Pneumoperitoneum protects the secretory and motor function of the intestine from extraabdominal factors. The good effects may result from a combination of mechanical, neuromuscular and vascular influences.

Tuberculosis of the intestines is frequent, affecting any region, mostly the ileocecal. The symptoms are: aversion to food; fullness after eating small meals;

sour eructations; nausea; occasionally constipation; abdominal pain aggravated by eating, relieved by fasting; rapid loss of weight, and local tenderness. In some instances a profuse diarrhea appears which precedes death. The stools are usually foul and when the lacteals are involved they are fatty.

The salutary results of pneumoperitoneum in tuberculous enteritis are notable. Because of the increased intraperitoneal pressure exerted on the sympathetic nerves the motility of the intestinal tract is inhibited, and for the same reason, the diarrhea, including the abdominal distress, is relieved, and the appetite augmented. There is a general amelioration of disagreeable digestive symptoms, particularly the abdominal pain.

Pneumoperitoneum reacts favorably on the stomach. The gastric motility becomes normal or improves, and acidity increases. Such symptoms as capricious appetite, anorexia, heartburn, postprandial pain, and hunger pain are greatly or completely relieved. As a result there is an increase in weight and relief of nausea and vomiting. In early cases the origin of gastrointestinal disturbance has been attributed to toxins causing reflex functional changes in the stomach such as hyper- or hypochlorhydria.

Gastrointestinal disturbances occasionally follow phrenic nerve interruption, and thoracoplasty, as: indigestion; difficulty in swallowing; belching, and intestinal stenosis due to kinking. Left phrenic interruption may be followed by loss of appetite; fullness without eating, and prolonged vomiting. Some patients develop gastrocardiac complexes, as precordial oppression, pain radiating to the left arm, respiratory distress, palpitation and extrasystoles. Here, this procedure was of benefit.

Pneumoperitoneum has tremendous therapeutic values proven by incontestable clinical satistics. Still it is not a panacea or an act of scientific perfection. It has its contraindications, draw-backs, and complications. It is fortunate that the major disadvantages are practically rare, and the minor infrequent.

To begin with, there are some patients who cannot tolerate the treatment because of pain, discomfort, and for various other causes. Many are plagued by constant abdominal tension, tightness, substernal soreness, belching, palpitation, dyspnea and nausea. Many complain of loss of appetite and dyspnea after the induction of "belly air".

The element of pain merits first analysis. It may be in the shoulder hypochondriac or epigastric areas. It may rest in the lower abdomen if it occurs during menstruation. Pain of severe character frequently settles in the back. The pain may result from stretching and irritation of the phrenic and vagi nerve endings. Torsion and pressure upon the abdominal blood vessels and mesenteric attachments may also produce pain. Generally, the pain may be a logical sequence of morphologic and functional alterations of the viscera which requires gradual accommodation. The pain may recur after each refill.

The stomach is attached by peritoneal folds to the liver, transverse colon, and pancreas. Following pneumoperitoneum a certain degree of gastroptosis can be observed, caused by the pulling upon the fixed attachments at its esophageal extremity. For the same mechanical action enteroptosis may result. There may be the formation of new herniations or exaggeration of old ones. Rectal hemorrhages may develop without ascertaining the cause. Hemorrhoids may appear as new formations, or the enlargement of those pre-existing. The condition of the peritoneum after pneumoperitoneum does not reveal any pathological changes. Very infrequently opaqueness, fibrinous deposits, adhesions, pseudoperitonitis and fluid formation are noted. The adhesions may produce various degrees of strangulation, kinking, and obstruction. The occurrence of ascites may be associated with fever, abdominal pain, nausea, vomiting, intraabdominal pneumocele, and omental adhesions. The occurrence of jaundice is rare.

Bobrowitz, in a very recent resume of 37 cases, concludes that approximately 60 per cent of the patients lost weight, had emaciation, abdominal distress, belching, nausea, occasional severe pain, and gastrointestinal disturbance. X-ray studies, two days before giving air, and repeated follow-up reveal elongation and narrowing of the cardiac portion of the stomach, widening or dilatation of the fundus, anterior displacement and drop of the stomach. These changes varied in hyposthenic, hypersthenic, and sthenic individuals. These symptomatic findings are not in accord with other phthisiologists.

The number of patients treated with pneumoperitoneum in the course of years runs into millions. The cases that were benefited are many. It would be useless to cite individual good effects, but, it is more significant to jot down the unfavorable aspects of pneumoperitoneum, which, compared to the above mentioned are real rarities.

Case Reports

Case 1:—Mrs. F. D. (Texas), white, 32, married, ill one year, sputum Gaffky III, with productive infiltration and apical cavity left lung. There was an infiltration at the second rib anterior. A left phrenic crush was done resulting in a 3 inch elevation of the diaphragm. In May 1951 a pneumoperitoneum was induced. The next day she developed 102 fever, abdominal pain and tenderness, vomiting, distention, pulse 120, and naausea. A Wangensteen tube was inserted but became clogged by a white thick gastric material. The stomach was washed out with warm saline, and the tube was reinserted, and left the entire night until 7:00 A.M. She became pale, and developed air hunger. The red cell count was 2,000,000; hemoglobin, 35 per cent. Several transfusions were given; also intravenous glucose and saline. A flat plate of the abdomen revealed fluid. The patient also developed a consolidation in the left base, which was considered pneumonia. A subsequent x-ray of the gastrointestinal tract was negative. It was concluded that she developed intraperitoneal hemorrhage and shock, and infection following pneumoperitoneum.

Case 2:—E. H., colored male (N. Y.), age 48, admitted from another sanatorium, with a diagnosis of chronic pulmonary tuberculosis. M.A. Sputum concentrations and cultures were negative since March 19, 1951. Pneumoperitoneum was induced June 30, 1950. X-ray revealed productive infiltration at the apex and anterior first interspace with exaggeration of the bronchovascular markings. The left lung was essentially negative. The patient constantly complained of abdominal pain and constipation. On September 8, 1951, he developed an episode of acute abdominal pain which was relieved by enemas, gastric lavage and morphia. Three weeks later he had another episode of abdominal pain which could not be relieved easily. He was sent to a general hospital, and after staying there a few days he felt better. The surgeon refused to operate. A gastrointestinal series revealed a narrowing in the large intestine. Shortly, the same condition returned with symptoms of partial intestinal obstruction. He was again sent to a general hospital. The surgeon stated that he would wait until the patient became more comfortable before he would attempt a laparotomy. Another gastrointestinal series revealed a small bowel obstruction at the jejunum and ileum with distention of the proximal two-thirds of the jejunum. The stomach was normal. The patient felt better for two weeks, and then the symptoms returned with greater severity. His blood revealed a moderate leucocytosis. The electrocardiograph consisted of rate 90, left axis deviation, sinus rhythm, T-2 flattened, T-3 inverted. Such changes are frequently found following pneumoperitoneum. The last x-ray report disclosed a narrowing of the jejunum at the junction of the ileum. There were multiple fluid levels within the bowel itself. He was sent to Bellevue Hospital where he was operated. It was found that the peritoneum was thick, and formidable adhesions pinching and enveloping the small intestine. He made a brisk recovery, gaining seven pounds before his discharge. This case demonstrates the formation of adhesions due to peritoneal irritation following pneumoperitoneum.

Case 3:—R. C., age 42, colored, admitted November 15, 1951. A positive sputum was found at Fordham Hospital July 30, 1951, where the x-ray demonstrated infiltration of the right upper lobe. He received streptomycin and P.A.S. He was transferred to a sanatorium as a case of pulmonary tuberculosis Grade II. He was given a second course of streptomycin and P.A.S. X-ray showed moderate bronchiectasis of both lungs. The laboratory reports: Sputum Gaffky, 8, 6, 3, 4; sedimentation rate 9-18; blood count normal; urine negative; Mazzini plus two. Pneumoperitoneum was induced in December 1951. He began to experience abdominal pain soon after each refill of 800 c.c. After the last refill of 1,000 c.c., his pain became very strong and localized at the right mid-abodmen on deep palpation. There was a leucocytosis of 12,000. The temperature ranged 101, 102, 103. On suspicion of acute appendicitis, he was sent to the hospital where an appendectomy was performed. The appendix was moderately inflamed. There was also some fluid in the abdomen. On his return, some residual pain remained, although in a lesser degree.

The three mentioned cases illustrate the complications that may arise during the administration of pneumoperitoneum. It is noted that the adverse symptoms are gastrointestinal, which appeared soon or very late after the treatment. Of course, this therapeusis is far from being innocuous, but the adverse effects are eminently rare.

In sifting over numerous protocols, and observing thousands of patients under active treatment, a rough estimate can be drawn of the effect of pneumoperitoneum upon the digestive apparatus. The cases that remain asymptomatic after treatment; those presumably improved; and those actually ameliorated, are about 95 per cent. Possibly five per cent constitutes those that cannot tolerate this procedure, and those who develop unfavorable signs and symptoms mentioned in the discussion. It is regretful, that pneumoperitoneum is limited within the confines of institutions that treat phthisis. It should be applied more extensively in the treatment of gastrointestinal ailments. It is a superb remedy. Nature supplies the medicament in inexhaustible and infinite volume.

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AN EVALUATION OF PRANTAL® IN ULCER MANAGEMENT

Follow-up of Preliminary Report

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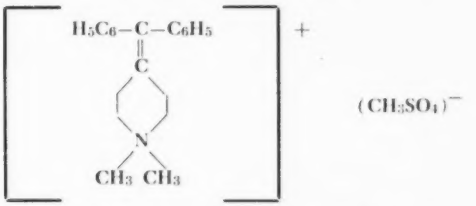
Bloomfield, N. J.

Preliminary report[1] was made of data on 26 peptic ulcer cases (23 duodenal, 3 gastric) treated with a new cholinergic blocking agent, Prantal Methylsulfate. Results in this preliminary study encouraged extension of the series to over 100 patients. The average patient in this present series has been observed from 18 to 24 months.

Chemically, Prantal is a quaternary ammonium (piperidylidene) derivative:



N,N-dimethyl-4-piperidylidene-1, 1-diphenylmethane methylsulfate

Pharmacologic studies[2-4] first disclosed the anticholinergic properties of this compound. The antisecretory effect of Prantal was studied in dogs, with marked reduction in the normal flow of gastric juice, as well as histamine-induced gastric secretions. There was impressive reduction in free and total acid secretion. Oral and intramuscular Prantal caused marked delay in gastric emptying time in dogs. Mydriatic activity was assayed in rabbits. Topical application of an 0.1 per cent solution of methantheline bromide caused mydriasis. Prantal in 10 times this dosage did not result in dilatation of the pupil. Extensive toxicity studies in several species indicated the high safety factor of Prantal.

Method

One hundred and seven peptic ulcer patients have been treated with Prantal Methylsulfate* since May 1951. Of this group 93 had duodenal ulcers, 14 had gastric ulcers. There were 91 males and 16 females whose average age was 44.9 years, ranging from 22 to 79 years. Ulcers had been present for varying periods of time from a few weeks to 30 years.

Patients presented symptoms of pain, nausea, belching, vomiting, hemor-

*Prantal Methylsulfate in three forms: Regular Tablets 100 mg., Repeat Action Tablets 100 mg., Injection 25 mg./c.c., was kindly supplied through the courtesy of the Clinical Research Division of Schering Corporation, Bloomfield, New Jersey.

rhage, hematemesis, melena, pyrosis, epigastric tenderness, hyperacidity and the usual signs and symptoms of ulcer.

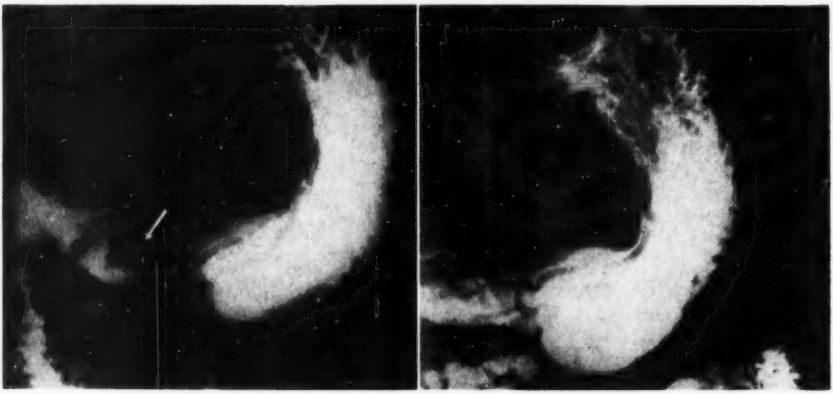


Fig. 1 Fig. 2

Fig. 1—Case 101 before Prantal.
Fig. 2—Case 101 after Prantal.

Eighteen patients had previously received therapy with other anticholinergics. Among them, 10 had previously had little or no relief from ulcer symp-

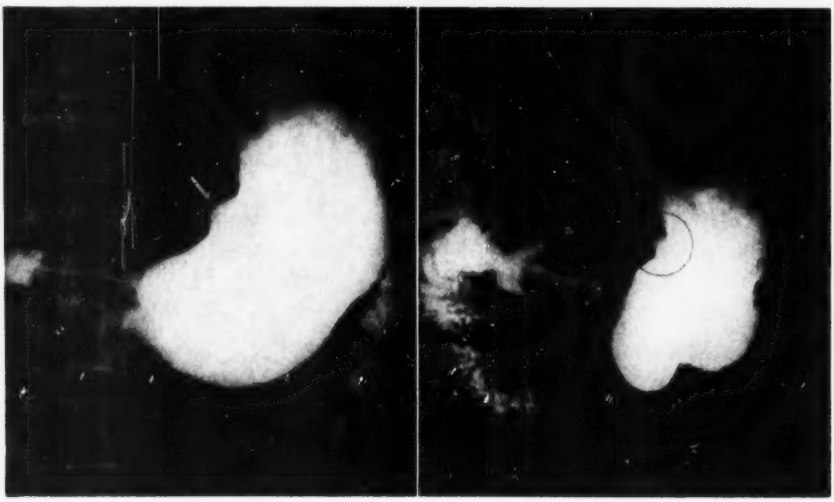


Fig. 3 Fig. 4

Fig. 3—Case 103 before Prantal.
Fig. 4—Case 103 after 2 weeks Prantal. Ulcer one-half size.

toms, even though treatment in some instances had continued for many months. Twelve had been unable to tolerate other anticholinergic drugs which caused

troublesome atropine-like side actions including severe constipation, xerostomia, urinary retention, blurring of vision, mydriasis and headache.

Our regimen consisted of Prantal in dosage of ½ tablet (50 mg.) to 2 tablets (200 mg.) q.i.d. Early studies were performed with the lower dosage. Increasing this dosage to a total of 800 mg. daily gives better therapeutic results and few troublesome side-effects.

All ulcers were x-rayed for confirmation of diagnoses and the majority were x-rayed again for evidence of ulcer healing.

Twenty patients were treated with Prantal alone, without dietary restrictions. Twenty patients were treated with Prantal and a bland diet. The balance of 67 patients were managed with Prantal, regular ulcer diets and antacids.

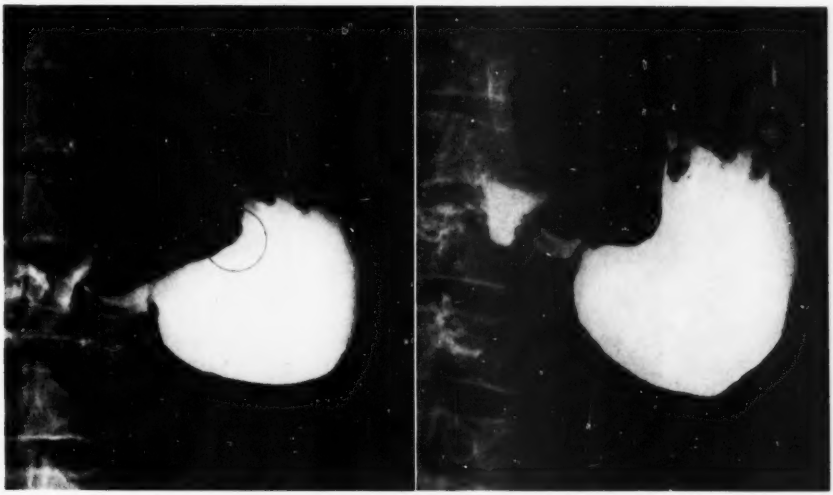


Fig. 5 Fig. 6

Fig. 5—Case 103 after 6 weeks Prantal. Ulcer one-quarter size.
Fig. 6—Case 103 after 4 months Prantal. No ulcer.

One patient with duodenal ulcer was hospitalized and treated with intramuscular Prantal Injection, 35 mg. q.i.d. Complete pain relief occurred within 24 hours. This regimen was maintained for 3 days, during which nothing by mouth was allowed. Nutrition and fluid balance were maintained by daily infusions and vitamins. Oral therapy was then instituted and the ulcer niche had disappeared when the patient was x-rayed 5 weeks later.

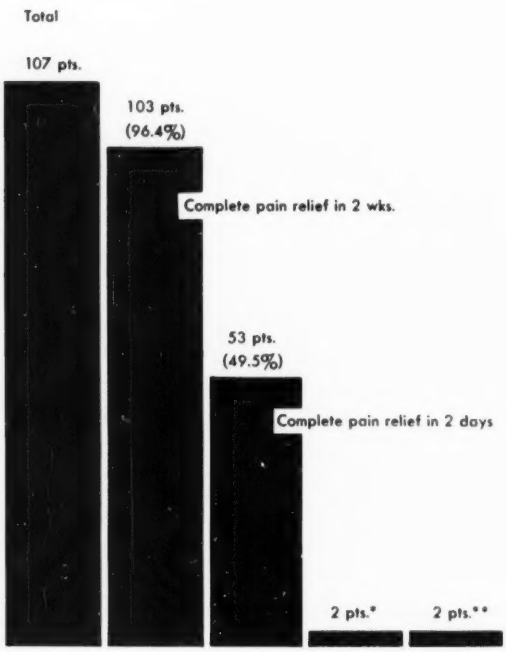
In a series of 5 additional patients, 25 mg. of Prantal Injection was given intramuscularly after determination of fasting free hydrochloric acid. Following injection, hydrochloric acid was determined at intervals of 15, 30, and 45 min-

utes (See Chart III). Progressive decrease of gastric acidity occurred in all patients.

We have recently employed a convenient dosage form—Repeat Action Prantal Tablets. These tablets consist of an enteric coated inner core of 50 mg. of Prantal surrounded by an outer layer of 50 mg. of Prantal available for imme-

RESULTS — CHART I

RAPID PAIN RELIEF FOLLOWING PRANTAL THERAPY



Questionable:

* 2 patients took medication spasmodically, therefore pain relief was not complete.

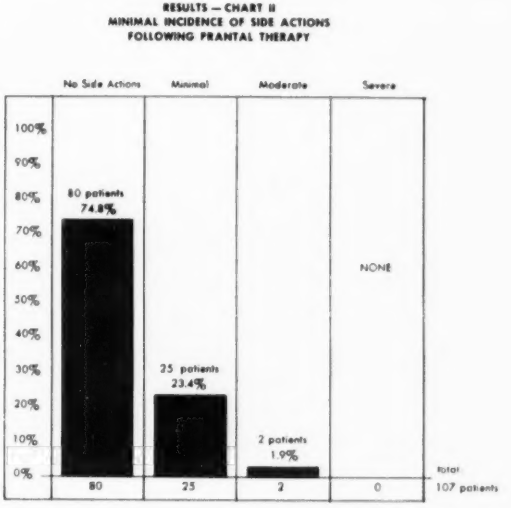
**2 patients experienced no relief. One of these was an alcoholic-psychoneurotic.

diate absorption. After 4-6 hours the inner core disintegrates, releasing an additional 50 mg. of the compound. This offers obvious advantages in that it may be ingested at bedtime and insures sleep uninterrupted by pain or medication. As a maintenance dosage, we employed Repeat Action Prantal—2 tablets q. 6 h.

Case Reports

Case 101:—W. L. 55 year old male with epigastric pain and tarry stools. X-ray and gastroscopy showed large antral ulcer on lesser curvature of the stomach and antral gastritis. Prantal, 200 mg. q.i.d., relieved pain in 3 days. Tarry stools ceased after 5 days. Repeat x-ray at 6 weeks showed no ulcer and only mild gastritis.

Case 103:—E. C. 54 year old male with epigastric pain of six weeks' duration. X-ray and gastroscopy showed huge gastric ulcer on the lesser curvature, with antral gastritis. Therapy: 200 mg. Prantal q.i.d. Ulcer in 2 weeks was reduced to one-half original size and in 6 weeks was reduced to one-quarter original size. After 4 months, follow-up x-ray showed no ulcer.



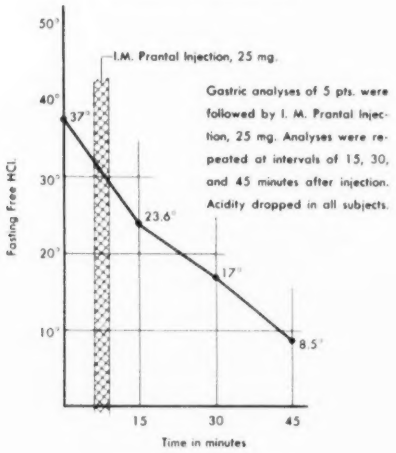
Case 106:—J. T. 59 year old male with epigastric pain for 2 weeks. X-ray showed duodenal ulcer. Prantal regimen, 200 mg. q.i.d. resulted in complete pain relief in 3 days. After 2 weeks, the ulcer was one-quarter its original size. X-ray at 6 weeks showed no ulcer.

Case 107:—P. S. 46 year old male with duodenal ulcer. Prantal 200 mg. q.i.d. resulted in complete relief from all symptoms in one week. Ten weeks later, follow-up x-ray showed no ulcer.

In contrast to the 12 patients who had been unable to tolerate other anticholinergic drugs, Prantal therapy has been remarkably effective in relieving ulcer symptoms and has caused minimal side actions. When side actions did occur, they were as a rule extremely mild and were usually mentioned by the patients only after questioning. In our entire series, 2 patients had a degree of side actions

which we have termed moderate. None had severe side actions. In no case was withdrawal of the drug necessary.

RESULTS — CHART III
REDUCTION OF GASTRIC ACIDITY FOLLOWING INTRAMUSCULAR PRANTAL
(AVERAGE OF 5 PATIENTS)



RESULTS–CHART IV

Ulcer Healing as Demonstrated by X-ray Following Prantal Therapy

Patients:	80 patients with gastric, and duodenal ulcers	
Dosage:	50 to 200 mg. q.i.d. Prantal Tablets	
X-ray Results:	Complete Ulcer Healing ..... (scarring present in 32)	74
	Improving	5
	Failure*	1
	Total	80
No X-ray Follow-up yet Available		27
		107 patients

*Alcoholic-psychoneurotic. Followed virtually no instructions for more than a week. Went to surgery.

Follow-up x-rays after a short period of Prantal therapy revealed less hypermotility, hyperirritability and pylorospasm than seen when initial x-rays were taken.

Diet Results:—None of the patients were permitted to smoke or to have alcoholic beverages. Control of these factors is at best somewhat dubious.

The previously mentioned 20 patients who received Prantal alone, with no dietary restrictions, responded excellently to this management. All experienced rapid pain relief and early healing.

Twenty patients on Prantal and bland diet fared as well.

The balance of 67 patients on Prantal, regular ulcer diet and antacids showed no significant difference from the above two groups. All had relief from belching and flatulence.

Ulcer Recurrences:—Recurrence of ulcer signs and symptoms occurred in 8 patients in this series. Generally, these patients had experienced periods of heightened emotional stress and tension (deaths or severe illnesses in the family, drug reactions to aspirin, etc.) Prantal therapy was reinstituted and all of these patients had rapid pain relief. Recurrence of ulcer symptoms was also relieved in 2 patients who were on a small maintenance dose of Prantal, by increasing the dosage. We now instruct all patients on maintenance doses to increase their

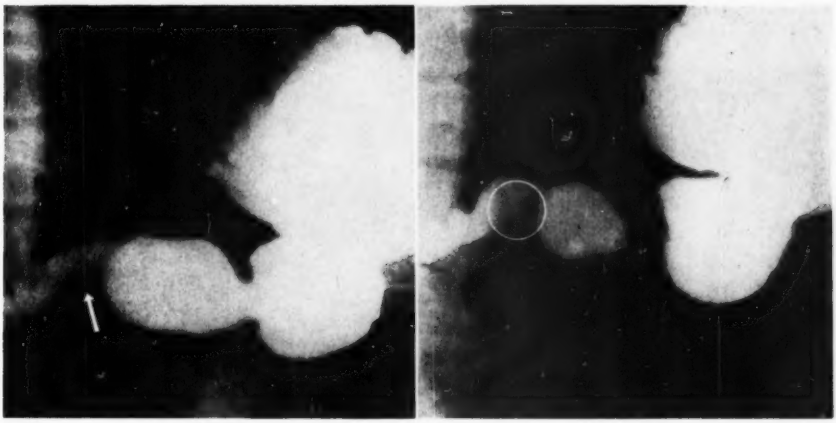


Fig. 7 Fig. 8

Fig. 7—Case 106 before Prantal.
Fig. 8—Case 106 after 2 weeks Prantal. Ulcer one-quarter original size.

dosage level when periods of stress occur. If they are no longer taking Prantal, we instruct them to employ a low maintenance dosage until the stress period has passed.

One patient in our original series had been successfully treated with Prantal following a severe episode of melena. Seven months following discontinuance of maintenance dosage, he experienced another hemorrhage. Prantal therapy gave dramatic relief. Several months later, again following discontinuance of Prantal, hemorrhage recurred for the third time. He was immediately placed on Prantal therapy and 2 days later was operated upon. At operation, his ulcer was found to be almost completely healed and a small eroded blood vessel was noted in the ulcer crater. It may be assumed that had this patient been maintained on Prantal therapy this ulcer would have healed completely. Since, however, this was the third hemorrhagic episode in this individual, operation was considered desirable.

Discussion

Anticholinergic drugs act by decreasing gastric acidity and motility[5]. Prantal acts by selectively inhibiting synaptic transmission over ganglia associated with gastric secretion and motility, without producing undesirable side-effects[1-10].

Before the advent of Prantal, occurrence of distressing atropine-like side actions with other anticholinergics was in such order as frequently to necessitate withdrawal of medication. Due to the selective action of Prantal, side actions are relatively infrequent. When they do occur they are of such mildness as rarely to necessitate withdrawal of the drug[1,5,7,9].

In a study by Hawkins, Margolin and Thompson[11] of gastric emptying time in normal subjects, Prantal was compared with methantheline bromide and

Fig. 9–Case 106. No ulcer after 6 weeks Prantal therapy.

placebos . . . "study shows clearly the oral efficacy of Prantal in delaying gastric emptying time . . . has less effect on small bowel motility than methantheline bromide, an observation in accord with 'specificity of action' claimed for Prantal".

Hoffman[12] treated 124 patients with advanced or complicated peptic ulcer. Many of these patients had shown little or no response to various rigid dietary regimens or medications, while others had responded but had had recurrences under treatment. One hundred and seventeen responded favorably to a Prantal regimen; operations were performed after medical therapy in 7 patients with surgical indications. Eighty of the patients were healed radiographically, 14 showed satisfactory but not yet complete healing, and the balance had yet to be examined.

Marks[8] summarizes his series as follows: "A new anticholinergic drug with minimal side-effects, Prantal Methylsulfate, was used in the clinical treatment of 46 cases of peptic ulcer. Eighty-nine per cent of these cases reported relief of pain within an average of 6 days. Eleven per cent of the patients on this dosage (50 mg. q. 5 h.) of Prantal were not benefited. Studies now being carried out suggest that some of the 5 failures might have been benefited with larger doses".

This is in accord with our experience in the present series. Larger dosage gives better therapeutic response without troublesome, atropine-like side actions.

Rowe, Grimson and Flowe[6], using Prantal Injection, reported: "Intramuscular injection of Prantal in 25-50 mg. amounts . . . reduced volume and acidity of gastric secretions in each of 9 patients tested. The effect equaled that occurring with Banthine in the usual test dose employed for this drug, 100 mg."

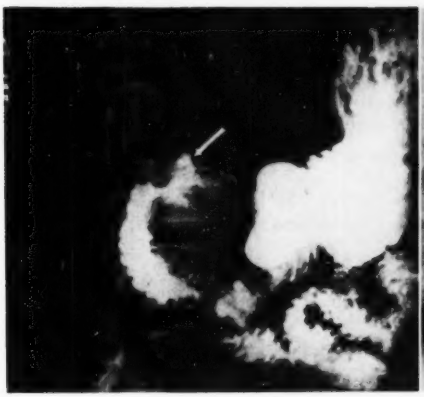


Fig. 10

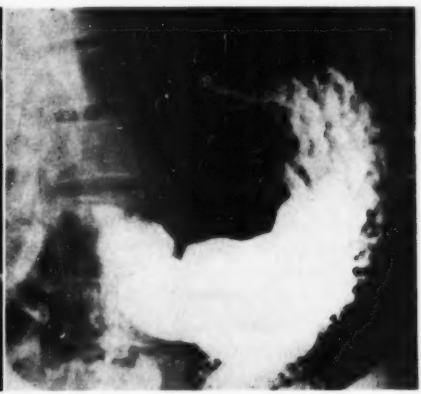


Fig. 11

Fig. 10—Case 107 before Prantal.
Fig. 11—Case 107 after 10 weeks Prantal therapy. No ulcer.

Zimmer et al. in a study[13] of the effect of Prantal upon gastric motility and secretion in the human concluded that the drug: effectively controlled the symptoms of peptic ulcer when given parenterally or in larger oral dosage; inhibited gastric motility given parenterally or in larger oral dosage and found that side-effects were not common in their series, and when present, to have been mild.

This is in accord with our experience with Prantal Injection in 5 patients, as recorded in Chart III. Further, Prantal Injection offers the ideal method of administration for patients in acute phase with hemorrhage, hematemesis or melena. The presence of blood in the gastrointestinal tract may interfere with absorption of oral medication. Medication, also, may be lost through vomiting. Intramuscular administration obviates these difficulties and results in prompt, effective therapeutic levels.

Much clinical evidence now exists as to the effectiveness of Prantal and its low incidence of severe atropine-like side actions. With other anticholinergic drugs, multiple side actions are frequent and interruption of therapy has been necessary in up to 20 per cent of cases[14].

In contrast, Prantal in our series has never resulted in side actions of more than moderate intensity. In no case was it necessary to interrupt therapy.

In the author's preliminary report[1] of 26 patients given 50 mg. Prantal q.i.d. . . . "no side-effects occurred in 25 patients". In a later report by the author[15], 21 patients given 50 mg. q.i.d. exhibited no side actions. Of 36 given 100 mg. q.i.d., 24 exhibited no side actions, 6 had single mild side actions, 3 had multiple mild side actions. Of 10 patients given 200 mg. q.i.d., 6 were without side actions. It is of interest to note that incidence of side actions did not increase significantly following doubling of the dosage q.i.d. in the group of 10 patients. This possibly demonstrates the vagotonia which is said to exist in peptic ulcer patients, predisposing them to toleration of anticholinergic drugs[16].

Rowe, Grimson and Flowe[6] reporting on a series of 44 patients whose regimen was 200 mg. q.i.d. said, "Prantal orally may produce a minimum degree of mydriasis and dryness of the mouth not considered troublesome by patients".

Vogel[5] in a preliminary report on 15 patients stated: "Undesirable side-effects were rarely observed. In no case was it necessary to discontinue the drug because of side actions".

Wissmer[10] makes particular remark of the contrast among other anticholinergics and Prantal in the matter of side-effects: "*On peut souligner la parfaite tolérance du produit qui, contrairement aux autres médicaments utilisés jusqu'à ce jour, n'a provoqué aucun trouble secondaire (mydriase, sécheresse de la bouche, etc.) chez nos 30 sujets*". [One can emphasize the perfect toleration to this product which, in contrast to other medications employed up to this time, has not produced any side-effects (mydriasis, dryness of the mouth, etc.) in our thirty subjects.]

Marks[8] concluded his paper by saying, "Side actions were minimal and in no case severe enough to necessitate discontinuing the drug. No urinary difficulties were encountered".

Summary

Data are presented on 107 patients with peptic ulcer treated with Prantal Methylsulfate in three forms: regular tablets, repeat action tablets and injection, with the following results:

Pain Relief:—Of 107 patients treated, 103 (96.4 per cent) experienced complete pain relief. Two patients took medication spasmodically and had incomplete pain relief. Two had no relief.

Healing:—Follow-up x-rays were taken of 80 patients. No x-rays are yet available of 27 patients. Of 80 patients 74 had complete ulcer healing (scarring was present in 32 with chronic peptic ulcer); 5 are improving; the one failure was an alcoholic-psychoneurotic.

Side Actions:—Of 107 patients, 80 had no side actions at all; 25 had minimal side actions, 2 had moderate side actions. None had severe side actions.

Conclusions

From clinical experience of over two years, pain relief data, absence of side actions and incidence of roentgenologically-proved ulcer healing, it is concluded that Prantal Methylsulfate is the drug of choice in therapy of peptic ulcer. Prantal offers the physician a safe, effective drug for therapy of gastric as well as duodenal ulcers. Prantal has the least side actions of any effective anticholinergic agent.

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CASE REPORT

Duodenal Ulcers (Multiple) In A Nine Year Old Boy Complicated by Pyloric Obstruction

H. M. ROBINSON, M.D.*

Brooklyn, N. Y.

J. B.:—In January, 1948, he presented the following history: For the past two years, since the age of eight years, he has been suffering with epigastric pains. He is the sixth child in a family of nine children. For two years he was treated in a general medical clinic with no definitive diagnosis. Examination in January, 1948 revealed a rather undernourished youngster of moderately sallow complexion. Heart and lungs were normal. There was tenderness in the epigastrium. Liver and spleen not palpable.

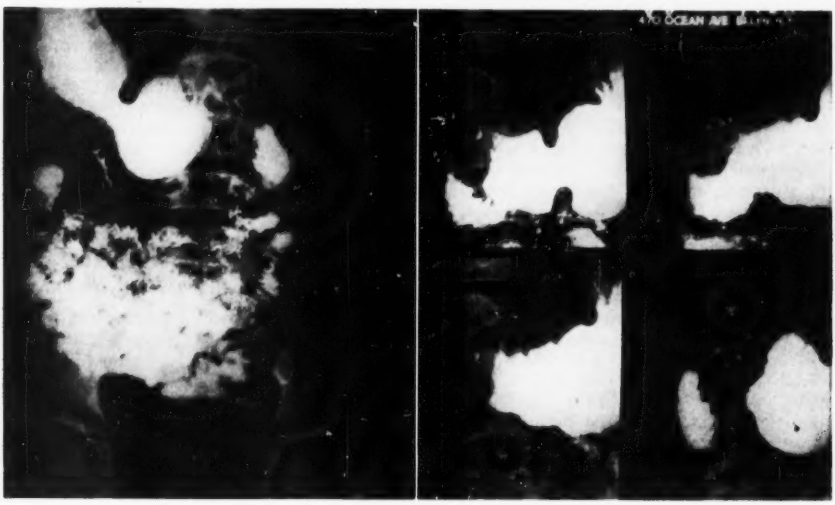

Fig. 1 Fig. 2

Fig. 1—J.B., 1948. Shows two niches within the duodenal bulb. No gastric retention.
Fig. 2—J.B., 1950. Persistence of ulcer niches within the duodenal bulb.

Radiographic examination of the gastrointestinal tract revealed the stomach normal. The duodenal bulb failed to fill properly and to empty completely, leaving two residual pockets of barium in the region of the duodenal bulb. These were interpreted as ulcer niches.

*Associate Gastroenterologist, Greenpoint Hospital; Associate Gastroenterologist, Jewish Hospital Chronic Diseases; Associate Attending Physician, Brooklyn Cancer Institute; Assistant Attending Physician, Maimonides Hospital.

The child was placed on an ulcer regime including Banthine and aluminum hydroxide gel. He responded fairly well to medical treatment and continued to go to school and carry on normal activities. An attempt to curtail his ball playing was not successful. In 1949 he returned again complaining of epigastric pain and he was again placed on rigid medical care. Since there were nine children in the family with a deceased head of the family, and limited financial support, I doubt that adequate care was given to the patient.

In 1950, another gastrointestinal series revealed a niche in the duodenal bulb with considerable gastric retention, 50 per cent in 24 hours. The child had lost ten pounds, was always tired and could no longer indulge in school and other activities. He was hospitalized at the Maimonides Hospital and surgery was not

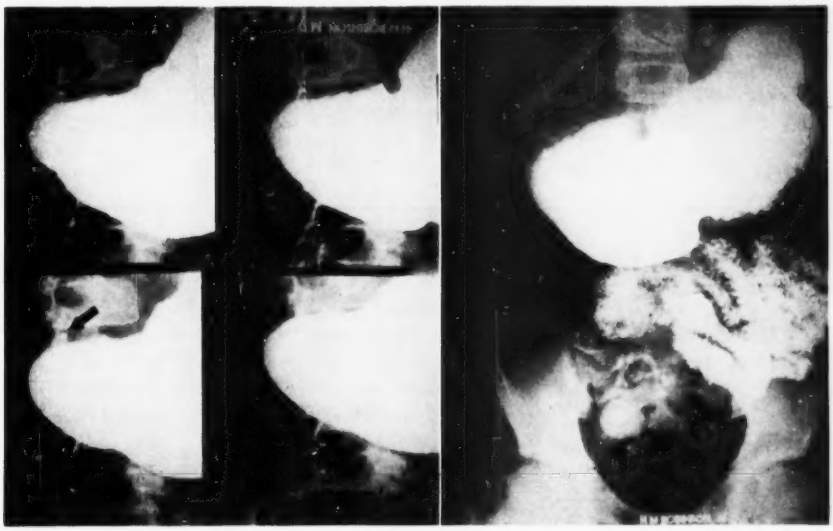

Fig. 3 Fig. 4

Fig. 3—J.B., Nov., 1951. Ulcer niche with gastric atony due to pyloric obstruction.
Fig. 4—J.B., 1952. Marked atony of the stomach with high degree of pyloric obstruction.

elected by the surgical staff. On medical regimen the degree of pyloric obstruction diminished as the acute stage of the ulcer subsided.

In 1951, the child returned complaining of nausea, vomiting and loss of weight. At this time, there was again 50 per cent retention and the child was again returned to the surgical service at the Maimonides Hospital.

Operation was performed in 1952, at which time a sub-total gastrectomy was done. At the operation, the duodenal ulcer was found penetrated into the bed of the pancreas. The ulcer was removed with pancreatic tissue. A pancreatic

fistula complicated his convalescence. Subsequent resection of the fistulous tract was done and uneventful recovery took place.

Conclusions:—This case report of a duodenal ulcer in a nine year old boy is presented because it is not common, although not rare. My impression is that if more children were studied radiographically, we would find a higher incidence of peptic ulcer in children.

Secondly, the two complications, penetration and obstruction developing so rapidly in a boy of nine with ulcer is rather a rare experience.

Thirdly, treatment of the complications should be identical with that in adults.

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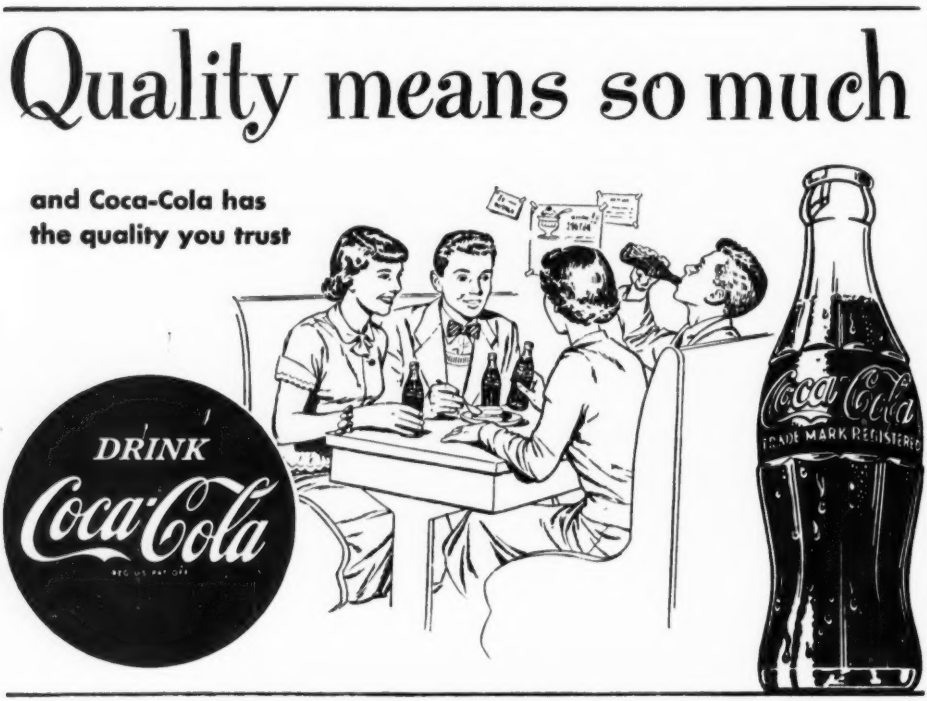

President's Message

On assuming the Presidency of the National Gastroenterological Association, our National Council has authorized a "President's Page", in our Journal.

It therefore gives me great pleasure to pay tribute to our founding fathers who have made our organization possible and who, by their ceaseless efforts, have contributed so much towards our success.

Time and space forbids anything but mere mention of their names, and there are undoubtedly some others whose names should be included, but I am mentioning those men with whom I personally have worked for the past several years.

First, of course comes the name of Anthony Bassler. We hope that his counsel and help will be ours for many years to come. Next we think of Roy Upham whose travels have been associated with our organization for so long. Then comes to mind Samuel Weiss, whose constant efforts with the THE REVIEW OF GASTROENTEROLOGY has carried our name far and wide, and with whose help we have, through the pages of our publication, reached all parts of the globe. Next we think of G. R. Manning who served so faithfully as president and then secretary for years. With him we recall Frank C. Yeomans whose counsel and help was always forthcoming in the early days of struggle. We could not conceive of our organization without our ever faithful treasurer, Elihu Katz, who has literally been the watch dog of the exchequer. We think also of William C. Jacobson, who has always been ready to serve in any capacity in which he is called upon. Then there are Frank A. Cummings, Harry Eberhard, Fred H. Voss, Henry Rafsky and A. X. Rossien. Here I must stop, realizing fully there are many others who have contributed much to the cause of the National Gastroenterological Association.

These men have done remarkably well under very trying circumstances. If we who follow in their footsteps do nearly as well for our organization, we shall surely have a wonderful future.

Sigurd W. Johnsen

EDITORIAL

Hygroscopic Gels

Hygroscopic gels as causes of esophageal obstruction are of more frequent occurrence than seem to appear in the literature. Self-medication upon advice of the commentator on the radio or television often results in near fatality unless recognized early and an attempt is made to disintegrate or dislodge the obstructing mass.

The most recent report is that of a patient whose esophagus became obstructed and the dramatic relief obtained after the ingestion of two teaspoonsful of liquid diastase. Any of the hygroscopic gels when taken with insufficient fluid, form an insoluble mass and may cause partial or complete closure of the esophagus.

I recall a patient who consulted me because of a roentgen diagnosis of complete obstruction of the lower end of his esophagus. His wife said that two nights previously he had eaten his usual dinner and did not complain of dysphagia, pain, nausea and hiccoughs. He was awakened about three o'clock in the morning, complaining of severe pressure in the upper abdomen and lower part of the chest, followed by nausea and hiccoughs. A physician who saw him within the hour diagnosed the attack as of coronary origin and administered a hypodermic of demerol. The patient fell asleep for about two hours and on awakening, again complained of severe pain. He tried to swallow some water without result. An electrocardiogram taken in the forenoon was negative. The physician referred him to a radiologist who fluoroscoped him and administered a barium mixture for visualizing the esophagus and stomach. The barium remained in the lower third of the esophagus, causing severe pain and pressure. Films taken showed complete obstruction. A report to that effect was sent to the referring physician, who advised the family that undoubtedly the patient had a neoplasm which required immediate attention.

The family, refusing to believe that this supposed neoplasm would suddenly become so urgent without previous symptoms, brought the patient, films and report to my office. On close questioning the wife recalled that her husband complained of constipation and, not obtaining relief from milk of magnesia, prune juice, etc., brought home a bottle of powder which a fellow-worker recommended on the say-so of the television commentator. Before retiring, he took several teaspoonsful and drank a few sips of water after swallowing the powder. This gave me the clue that the patient was a victim of ill-advised self-medication and lack of sufficient diluent.

Lavage of the esophagus dislodged a large amount of barium mixed with the mucilagenous gel. This lavage was continued and the patient was given two teaspoonsful of caripeptic liquid which is a combination of the enzymes of

carica papaya and malt diastase. Thirty minutes later, the patient was relieved of his distress and was able to swallow some water.

Re-x-ray of the esophagus two days later with a small amount of barium mixed with caripeptic liquid failed to show the presence of an obstruction.

Another patient of mine who swallowed a large bolus of unchewed food complained of pain and dysphagia, was given 6 grains of papain and half an ounce of extract of malt, repeated three times daily. After several doses, he was relieved of his distress.

Hygroscopic gels used for laxative purposes should be well diluted and additional glasses of water should be imbibed during the day, provided cardiovascular, hypertensive, prostatic, ascitic or other organic condition contraindicates the ingestion of large amounts of fluid. Care should be used in administering barium to patients where the history reveals that they have taken some form of a hygroscopic gel with little fluid.

The administration of liquid takadiastase, caripeptic liquid and/or extracts of malt, two to four teaspoonsful orally or through the duodenal tube, is capable of converting the carbohydrate content of these gels into water-soluble sugar, while papain converts proteins to amino acids in acid and neutral media.

Administration of one or the other of these preparations in achalasia with marked dilatation, in diverticula and in inoperable lesions of the esophagus, often gave temporary relief to some patients.

SAMUEL WEISS, M.D.

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NEWS NOTES

IVth European Congress of National Gastroenterological Associations

The IVth European Congress of National Gastroenterological Associations will take place in Paris, France from 27 June to 1 July 1954 inclusive. The President of the Congress is our Honorary Fellow, Dr. René Gutmann and the Secretary-General is Dr. André Busson.

Following the Congress in Paris, the delegates will go by special train to Vichy, France to attend the Congress of Hepatology from 3 to 5 July 1954. This Congress will be under the auspices of the Society of Hepatology of Vichy and there will be social activities as well as scientific sessions.

Dr. Roy Upham, Secretary-General of the National Gastroenterological Association, has been appointed chairman of the official National Gastroenterological Association delegation to the Congress and those interested in attending the sessions may get in touch with Dr. Upham at 45 E. 74th St., New York 21, N. Y.

Errata

Figures 3a and 3b of the article "Diseases of the Stomach and Duodenum" by Dr. Sol Fineman which appeared in our October 1953 issue were incorrectly printed on page 735. These figures should have appeared on page 716 of Dr. Fineman's article.

The illustrations which were printed on page 716 should have appeared on page 735 of the article "Evaluation of the Erythrocyte Sedimentation Rate in Gastrointestinal Diseases" by Dr. Emanuel Lipschutz.

We sincerely regret this error and corrections are being made in the reprints.

ANNOUNCEMENT

Effective with the January 1954 issue, the name of our publication will be changed to The American Journal of Gastroenterology.

Also on that date, the subscription price will be increased to $6.00 for one year and $10.00 for two years in the United States and its possessions. Foreign subscription rates will be $8.00 for one year and $12.00 for two years.

In Memoriam

We record with profound sorrow the recent passing of two of our Honorary Fellows:

Dr. Max Einhorn of New York, N. Y., the father of gastroenterology who passed away in his 90th year.

Dr. Horace W. Soper, Past-president of our Association, who died in St. Louis, Mo. in September, at the age of 86.

We extend our deepest sympathies to the members of the bereaved families.

BOOK REVIEWS FOR GASTROENTEROLOGISTS

RHEUMATIC DISEASES, DIAGNOSIS AND TREATMENT: Eugene F. Traut, M.D., F.A.C.P., Associate (Rush) Clinical Professor of Medicine, University of Illinois, Attending Physician to the Cook County Hospital and to the West Suburban Hospital, Oak Park, Illinois, Associate Attending Physician to the Presbyterian Hospital of Chicago, Director of the Arthritis Clinic of Cook County Hospital, etc. 942 pages with 192 illustrations. C. V. Mosby Co., St. Louis, Mo., 1952. Price $20.00.

This is the newest encyclopedic volume on the all important subject of "Rheumatic Diseases".

The reviewer enthusiastically recommends this textbook to all practicing physicians, internes, residents, fellows, teachers in all medical schools and to medical students. Understandingly and instructively, the author presents etiology, classification and pathogenesis of diseases of joints, psychologic factors (in rheumatoid arthritis), focal infection, "Collagen diseases" by Hans Popper and John H. Lunseth, experts in pathology and medical research.

Drs. Franz Alexander, A. Johnson and L. B. Shapiro contributed (chapter III) "psychologic factors". "Histopathology of muscle in rheumatoid arthritis", etc., was prepared by Kenneth M. Campione, M.D. and the author (chapter VII). The "ocular manifestations of rheumatic diseases" (chapter XIII) by Wm. F. Hughes, Jr. Dr. Fred W. Park discusses the prevention of deformities in joint disease (chapter XLVII) and Dr. Fremond A. Chandler and Dr. Charles V. Heck present "the surgical treatment of arthritis" (chapter XLIX) in concise and adequate manner. "Roentgen therapy of arthritis" by Harry Slobodin, M.D. (chapter XLV), is interestingly instructive.

DISEASES OF THE HEART AND CIRCULATION: Albert A. Fitzgerald Peel, M.A., D.M. (Oxon.), F.R.F.P. (G), Physician for Diseases of the Heart, Victoria Infirmary, Glasgow Medical Consultant, Department of Health for Scotland and University of Labour and National Service Recruiting Boards, Formerly Professor of Medicine, Anderson College of Medicine, Glasgow, Former Visiting Physician E.M.S., Scotland. Second Edition. 472 pages. 1952. Price $7.50.

This improved up-to-date compact and instructive second edition of a practical small volume on diseases of the heart and circulation by an experienced cardiologist, will be helpful to medical students, general practitioners, residents, internes and young cardiologists.

This authoritative work can very helpfully and profitably be used by instructors and teachers of junior and senior medical students and those interested in cardiology.

The author satisfactorily and in brief but adequate manner, presents normal and morbid physiology and symptomatology and physical signs in cardiovascular disease.

Well discussed are cardiacrhythm, ventricular complexes in cardiograms, cardiac-vector and vector diagram pericarditis, endocarditis, arterial disease (pages 268-346), cardiovascular congenital anomalies, psychoneurosis, endocrinopathy and the heart in vitamin deficiency and anemias, etc. Ballistography receives only two small paragraphs (page 68)—a total of 16 lines.

The increasing use of ballistography by (Isaac Starr, Arthur M. Master and Associates, William Dock, Ignacio Chavez, Demitrio Sodipallares (Mexico), Traynor, H. R. Brown and Hoffman and deLalla, Porter, Chesky, Mathers and Nickerson, Makinson, Frankel and Rothermich, J. W. Gordon (1877), Henderson (1905), Rawson, H. A. Schroeder and Joseph and Starr, H. Mandelbaum, Caccese and Schrager and many others) many clinicians, cardiologists and investigators, has now made this form of investigation sufficiently important to deserve considerable discussion.

Electrocardiography, with all its modern improvements, roentgen studies, angiocardiography and other recently reported advanced forms of investigation of cardiovascular diseases and anomalies have added much valuable information in our studies of disorders of the circulation.

The reviewer recommends this useful compact work for quick review, for medical students, the beginners in cardiology and for general practitioners interested in cardiology.

THE MECHANISMS OF DISEASE—A STUDY OF THE AUTONOMIC NERVOUS SYSTEM, THE ENDOCRINE SYSTEM AND THE ELECTROLYTES IN THEIR RELATIONSHIP TO CLINICAL MEDICINE: Joseph Stambul, M.D., Chief of the Cardiovascular Department of Albert Einstein Medical Center (Eastern Division), Cardiologist to Deborah Sanatorium, Browns Mills, N. J. Foreword by George Morris Piersol, B.S., M.D., M.A.C.P., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania, Editor-in-Chief, The Cyclopedia of Medicine, Former Editor of the American Journal of Medical Sciences. 746 pages. Froben Press, Inc., New York, N. Y., 1952.

This most unusual book begins with an excellent and philosophically worded foreword by a distinguished Philadelphia physician and one of the early leaders with Alfred Stengel and O. H. Perry Pepper in the advancement and progress of the American College of Physicians—Dr. George Morris Piersol.

The reviewer was most favorably impressed with the many interestingly, instructive, unorthodox concepts presented by the industrious, most energetic and sincere author of this stimulating book. The reviewer gladly recommends this volume to all physicians, investigators, physiologists, gastroenterologists, psychiatrists and advanced medical students.

The author carefully and studiously tries to interpret the mechanisms of disease. He points out the way in which various tissue cells of the human body carry on their activities under normal circumstances and how they are affected by noxious agents of exogenous and/or endogenous origin causing disease. The author has worked most diligently and for over ten years, has studied much of the available literature for compilation in a most condensed form, some of his findings and conclusions in this excellent volume of 746 pages.

There is included an exhaustive bibliography of hundreds of references of 42 pages, also a subject index of 15 pages.

This most unusually informative book should be read by all teachers, instructors and advanced medical students, as well as all general practitioners, clinicians and specialists.

1952 YEAR BOOK OF GENERAL SURGERY: Edited by Evarts A. Graham, M.D., with a section on Anesthesia by Stuart C. Cullen, M.D. 670 pages, illustrated, cross index and authors index. The Year Book Publishers, Chicago, Ill., 1953. Price $6.00.

This volume also follows the pattern of previous year books on surgery.

Under the editorship of Dr. Graham, important items from medical journals dealing with diagnosis and treatment are abstracted and when deemed advisable, illustrations are also included.

An interesting item on page 351 is Lymphopenia in Diagnosis of the Acute Abdomen. Studies of white blood cell counts in a variety of acute conditions were made and Lymphopenia was expressed as absolute or relative. Lymphopenia is an unfavorable sign in the leucocyte picture and a return of the lymphocyte count toward normal suggests a good prognosis.

Another interesting abstract deals with Roentgen manifestations of Acute Intermittent Porphyria.

The section on Anesthesia should be of interest both to the surgeon and the anesthetist as many new and pertinent items are found in this section.

General as well as special surgeons will find the part on General Surgery of value.

1952 YEAR BOOK OF PATHOLOGY AND CLINICAL PATHOLOGY: Edited by Howard T. Karsner, M.D. and Arthur H. Sanford, M.D. 400 pages, illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1953. Price $5.50.

In reading this book on pathology and clinical pathology, the reviewer is amazed at the great amount of material which had to be read and the essentials abstracted. Unfortunately, the general practitioner is too busy to follow the newer trends in the scattered medical literature. In this volume, he will find comprehensive abstracts dealing with general and special problems in medicine, surgery and research.

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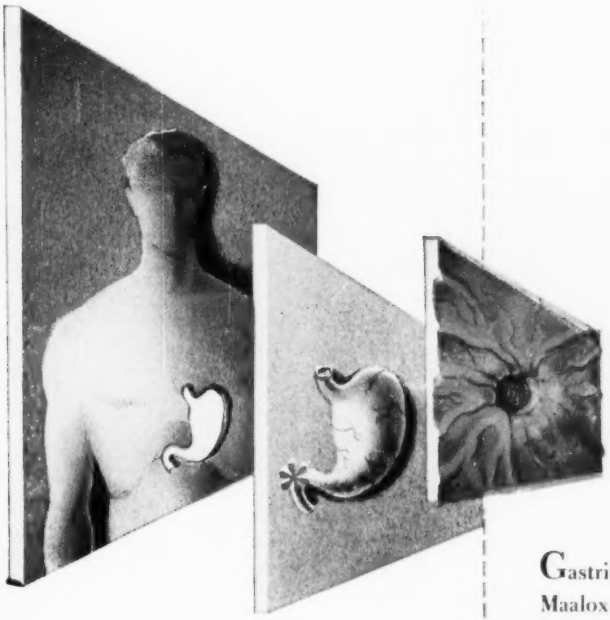

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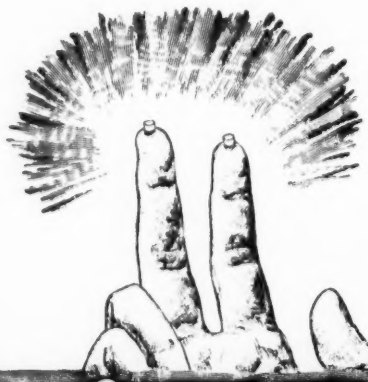

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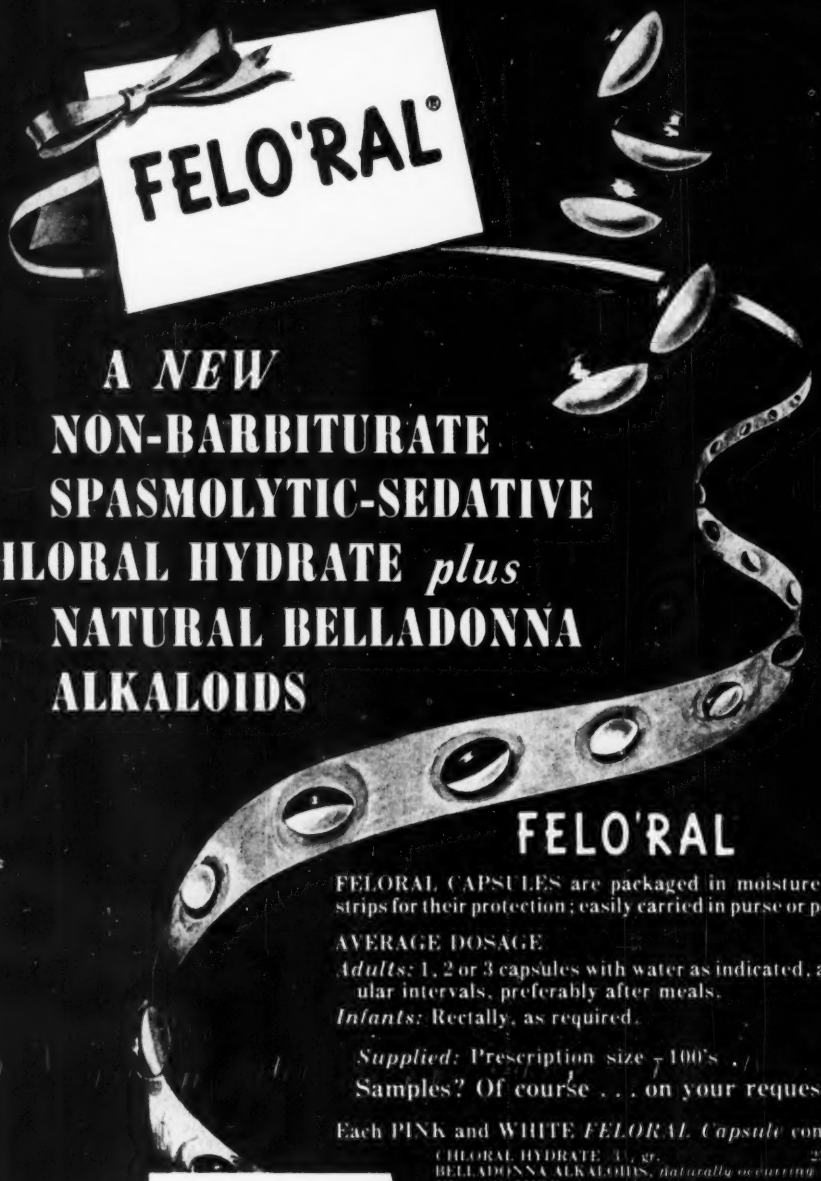

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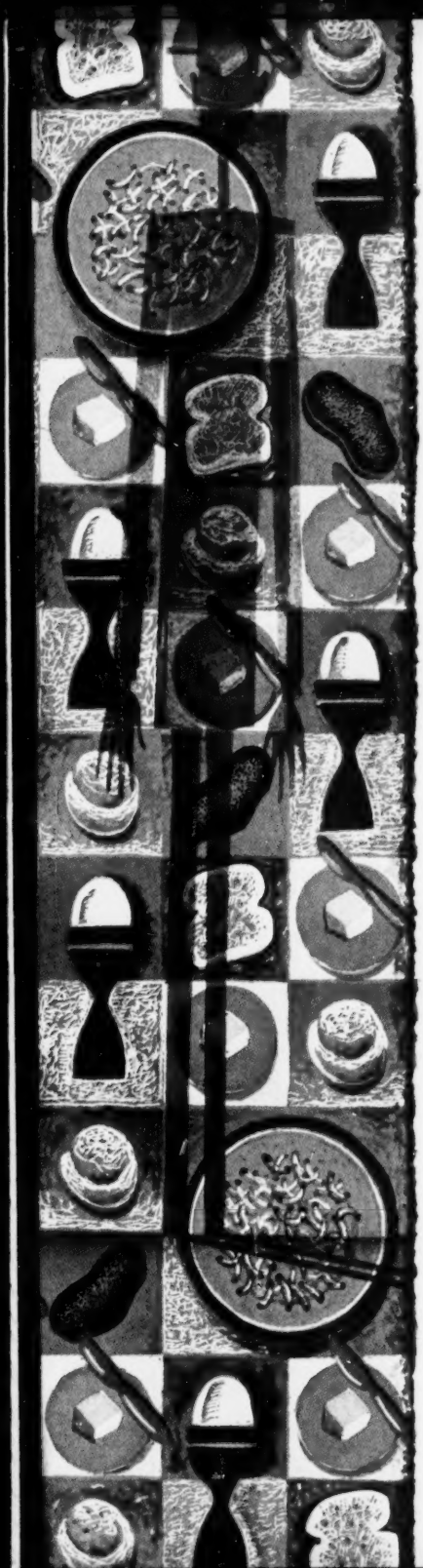

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